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VOLUME 3 *Chicago Number* NUMBER 6

THE MEDICAL CLINICS OF NORTH AMERICA

289826
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MAY, 1920

Index Number

PHILADELPHIA AND LONDON
W. B. SAUNDERS COMPANY

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PUBLISHED BI-MONTHLY (SIX NUMBERS A YEAR), BY W. B. SAUNDERS COMPANY, WEST WASHINGTON SQUARE, PHILADELPHIA
PRINTED IN AMERICA

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THE MEDICAL CLINICS
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VOLUME 3 NUMBER 6

CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

LETHARGIC ENCEPHALITIS

Features in History Shown; Discussion of the Pathology and Nature of Encephalitis; Etiology; Discussion of Symptomatology; Various Types of the Disease; Differential Diagnosis; the Course Taken by Lethargic Encephalitis; Treatment and Management; Prognosis.

THIS patient is a married woman, aged forty-three years, who was taken ill on or about January 4, 1920. She is unable to give a clear account of her first sensations, but states that she was extremely nervous and that very early she began to have double vision. This was followed by disturbance of gait, so that when she went through a door she was likely to bump on either side. It was not the typical gait of cerebellar ataxia, but was a general ataxia. As she expressed it, she "walked like a drunken sailor."

Another thing that early excited her attention and wonder was a choreiform movement of her upper extremities—when she tried to hold her arm out straight, it would suddenly jerk downward. Frequently she dropped things. Associated with these symptoms of nervousness, double vision, and chorea was a certain amount of nervous excitement which in the course of time passed into nocturnal delirium with hallucinosis. For example, when I first saw this patient on January 11th she said she had seen horses and wagons going through the air up in the corner

of her bedroom. She also complained of marked hyperacusis, saying that the slightest sounds were very loud.

Following this stage of prodromal symptoms, lasting from January 4th to 11th, there was a stage of extremely active symptomatology. Fever appeared the night of January 11th, being at first about 100° F., but subsequently rising to 104° and even 105° F. The hallucinations of vision became much more pronounced. The night of the 11th the delirium was extremely great, and was associated with marked restlessness, so that she

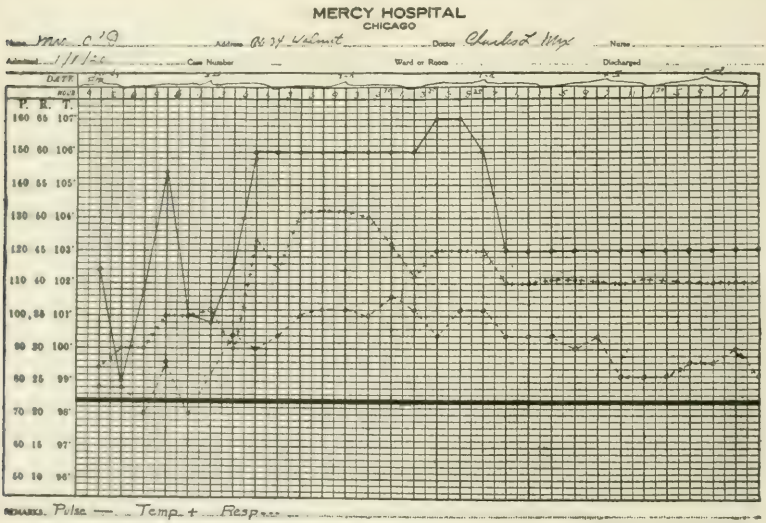

Fig. 249.

tried to get out of bed, out of the room, and even out of the window. She acted very much as a patient does in acute mania. The following day she passed into a stage of beginning lethargy. She would lie with half-closed eyes, whether or not due to an insufficiency of the levatores palpebrarum superiorum I do not know, but could be easily aroused. She would not always answer properly when spoken to, but usually betrayed no sign of active delirium. Sometimes the lethargy passed over into stupor, and when aroused from this condition she was usually disoriented as to time and place.

During the week between the 11th and 18th the fever remained high, but toward the end of the week the temperature subsided to 101° to 102° F. After the 18th the temperature dropped slowly still further, until finally on January 30th an all-day normal was reached. This was followed on the 31st by a slight rise of temperature, which persisted also on February 1st, but since that day she has had no fever.

The most striking feature in the case of this patient is the behavior of the pulse, which is illustrated by Fig. 249. It will be noted that the pulse ranged from 112 to 120 during January 11th and 12th, but that suddenly on the 13th, without any apparent reason, it jumped to 150. It will also be noted that the pulse remained at exactly 150 for nearly three days, the only variation being one individual instance, when it went up to 160. Having remained steadily at the exact level of 150, the rate dropped back rather abruptly to its old rate of between 100 to 120. There is a reason for this pulse behavior, which will subsequently be commented upon.

Physical examination discloses the following facts: On January 11th there was pupillary inequality, the right pupil being considerably larger than the left. There was, however, no irregularity of pupillary outline. Examination for sensory disturbances showed nothing but hypersensitiveness, though subjectively she complained previous to the time that she was first seen, during her week of prodromal symptomatology, of sharp neuritic pains in her right arm. Indeed, when first seen, she said that her trouble was "neuritis," because of the predominance of this symptom. Motor disturbances were also present, as choreiform jerks, but not in the form of paresis or paralysis of the extremities.

The only other changes noted were in the cranial nerves. There was a slight paresis of the right internal rectus with a mild degree of divergents trabismus, which accounted for her double vision. There was also a slight amount of facial paresis on the right side, due to involvement either of the nucleus or peripheral portion of the seventh nerve, since the right forehead could not be wrinkled as well as the left. The right nasolabial fold was partially smoothed out, and the right corner of the mouth,

on innervation, was shown to be paretic. In addition to this she had some disturbance of the motor supply of the twelfth nerve, manifested both by dysarthria and dysphagia. On testing out her labial letters, p-b-f-v, she did very well; her linguals and dentals, l-r-t-d-n, were not so well executed, owing to disturbance of the tongue. The gutturals, ch-k and g, were not pronounced well at all. She had herself noticed that she could not talk well and alluded to her dysarthria as "stammering." It really was not stammering, but was rather an indistinctness of speech. On another occasion she more aptly expressed it when she said she "talked as though her mouth was full of mush." The trouble with swallowing never reached a degree which made eating and drinking difficult, but it was manifest to an extent just sufficient to be annoying.

Examination of the other cranial nerves was negative with the exception of the nucleus ambiguus, which is the vagus inhibitory center. It will be noted by reference to the pulse chart that her pulse ranged at 150 for two and a half to three days. This marks the stage of paralysis of the vagus inhibition. When pneumogastric-cardiac inhibition is done away with the pulse usually jumps to 150 or 160. In the case of this patient we have almost an experiment in pathology. The chart very clearly shows the exact time at which cardiac inhibition became completely paralyzed, and the exact moment at which cardiac inhibition was restored to the patient. During the time that the pulse ranged at 150 it was perfectly rhythmic and equal. Moreover, the patient was not especially dyspneic. This is interesting, as demonstrating that the rapid pulse-rate was in no wise due to any cardiac defect other than the mere removal of cardiac inhibition.

Examination of her reflexes showed exaggeration of the knee- and ankle-jerks, of the bicipital and tricipital reflexes, and of the scapulohumeral reflex on each side. There was a left-sided Babinski reflex, which persisted for about a week, and then disappeared. The plantar reflexes were always somewhat reduced and the epigastric and hypogastric reflexes were absent.

Examination of the motor tracts showed no demonstrable

paresis, notwithstanding the presence of the left Babinski reflex. There was, however, marked static and locomotor ataxia. She could not stand with the eyes closed, showing the ordinary Romberg sign, and when she attempted to walk she showed a gait which reminded one of cerebellar ataxia. It is, however, an ataxia which may be said to be due to involvement of equilibration centers on both sides of the body. Possibly since the pyramidal tract on the left side, as shown by the Babinski reflex, was involved in some part of its course, it may be that the direct cerebellar tracts, or their extensions to the cerebellum, are involved and are responsible for the ataxia.

Aside from these changes involving the cranial nerves, the equilibration apparatus and the reflexes, she also showed signs suggesting a meningoradiculitis. By this we mean signs of disturbance of both meninges and posterior nerve roots. The meningeal signs are betrayed chiefly by her delirium and, during one night, by her acute mania. The radicular or root-signs are betrayed in her alleged brachial neuritis which consisted of rather severe pains which for the most part followed the ulnar nerve distribution. This neuritic pain began, not very severely, about January 7th, in the right arm, and two days later moved to the left arm. As the pains appeared in the left arm those in the right arm became less intense. In type the pains were distinctly shooting and stabbing, reminding one in a general sort of way of the lightning-pains of tabes.

Examination of the blood showed no noteworthy increase in leukocytes even when the temperature range was as high as 104° or 105° F. Two examinations were made, the first showing 7600 cells and the second 8700. The differential count was 71 per cent. polymorphonuclears; 24 per cent. small lymphocytes, 3 per cent. large mononuclears, and 2 per cent. polymorphic eosinophils. On the day after she was first seen a spinal puncture was made and the fluid was found to be under considerable pressure. A cell count was made within fifteen minutes after its removal, and showed 105 cells to the cubic millimeter. There was also a slight increase in globulin, shown by both the Ross-Jones and Pandy test. A differential count, unfortunately, was

not made of the spinal cells, so I am not able to say whether lymphocytes predominated or not.

Since the patient has been in the hospital she has passed through a stage of delirium, excitement, and mania, lasting only for a period of thirty-six hours, and a long period of lethargia. For several days she would lie with the eyes half-closed, apparently oblivious to everything that was going on, yet she could always be aroused. When spoken to she would answer, and usually to the point, but she never volunteered a single remark. She declared that she never slept, but the nurse in charge was equally positive that she slept all the time, because she would lie perfectly motionless for hours and would say nothing unless aroused. During the night lethargy at times gave way to mild delirium, so that she would mutter and talk quite a bit, more or less spontaneously, and sometimes in answer to questions by the nurse. In time, however, the nocturnal delirium ceased and lethargy and asthenia predominated. Once during the last week of January, when the nurse was temporarily out of the room, the patient attempted to stand, as she expressed it, "just to see if she could." She would have fallen if she had not suddenly taken hold of a chair. This weakness seems to have been more of an asthenia than a paresis or paralysis, since movement of all muscles is present.

I want to call your attention to another symptom present in some cases of encephalitis lethargica which this patient shows. During the fall I have seen 8 patients with encephalitis lethargica, including this patient, and 7 of them, during the period of lethargy, have shown catatonia beautifully. In fact, they behaved exactly as the catatonic type of dementia præcox. If you took hold of the arm, for example, and raised it to a certain position and then let go of it, the patient allowed the arm to remain in the position in which you had placed it indefinitely. Some observers have reported catalepsy in their cases, this being merely an extension of the degree of catatonia.

Discussion of the Pathology and Nature of Lethargic Encephalitis.—Not very many postmortem examinations have been performed on cases of lethargic encephalitis. A few have been

done in England, some in America, and some here in Chicago. Different observers have reported different findings, but the different findings reported are all similar in distribution and type, and apparently account for the symptomatology. Practically all observers report the finding of macroscopic and very small, pin-point hemorrhages scattered throughout the gray mater of the floor of the aqueduct of Sylvius, of the third ventricle, the fourth ventricle, and the posterior part of the pons and medulla, and the lenticular nuclei. Microscopic examination of these areas of miliary hemorrhages show that the small blood-vessels, especially the vasa vasorum, are infiltrated with lymphocytes and plasma cells in the adventitia. Scattered through the same areas are found foci of interstitial inflammation, the cells of which consist chiefly of neuroglia cells, lymphocytes, and polymorphonuclear leukocytes. These lesions clearly account for the involvement of the cranial nerves and for some of the symptoms of disturbance of the long tracts. One can easily see why certain observers insist that encephalitis lethargica is nothing but polioencephalitis superiora, or posteriora acuta, since in these diseases the anterior or posterior cranial nerve motor nuclei are the foci of involvement. It is easy to see, too, why some observers might classify lethargic encephalitis as a pontobulbar or a cerebellobulbar polioencephalitis. For example, if in a given case, as in one reported by Mills and Wilson, the involvement be almost wholly in the posterior portion of the brain stem, the anatomic diagnosis of cerebellobulbar polioencephalitis is amply justified, but in the case just mentioned lethargy was also present—and this symptom serves to bring the particular case mentioned into the general category of lethargic encephalitis.

From the pathology thus far mentioned it is also easy to see why cases, as a rule, almost always begin with double vision. The first sign is usually due to some involvement of one or more of the subnuclei of the third nerve immediately beneath the aqueduct of Sylvius, but in one case that I saw, a man of forty-two years, the initial symptom was a paresis of one of the trochlear nerves, the reduplicated image in this double vision being above and to one side of the true image. In all 8 cases which

I have seen double vision was one of the prominent initial symptoms.

Besides the involvement of the gray matter of the motor cranial nuclei just mentioned there is, in the majority of instances, another site of disturbance. Small miliary hemorrhages are apt to be found also in the basal nuclei, in the centrum ovale, and in the brain stem. It is very easy to understand how certain cases of Buzzard's were described by him as closely resembling acute paralysis agitans. If the disturbance is largely in the lenticular and caudate nuclei, we should expect a symptom-complex in which no ocular palsies occurred, but in which symptoms resembling those of Parkinson's disease were predominant. We should not be surprised at Buzzard's statement that a patient of his who had been ill only a few days presented a picture that one ordinarily finds after Parkinson's disease has persisted for about ten years. The mask-like, expressionless face, the rigid but not really paralyzed legs, the tremor, the posture, and the gait would necessarily closely resemble those of paralysis agitans, but, unlike paralysis agitans, there would be added the symptoms of asthenia and lethargy, the latter being especially suggestive of an associated midbrain lesion.

Not only does the central gray matter show marked involvement, but at times the leptomeninges and cerebral cortex are involved. Often the only thing found in the leptomeninges is a congestion of the blood-vessels, but at times cellular infiltration of the meninges is actually found on microscopic examination. Hence, it is not strange that some of these patients should show a predominance of maniacal symptomatology. Indeed, in some instances there are thromboses of small or middle-sized blood-vessels with small areas of softening, so that there is an actual meningo-encephalitis in some cases. Thus is explained the type of case in which the symptomatology is always general and without localizing symptoms.

What is the etiology of so curious a set of symptoms and pathologic findings? Those of us who have observed nervous diseases for a good many years cannot help being struck by the fact that a similar set of disturbances were described after the

epidemic of influenza of 1890, chiefly by the German observers, under the name of "epidemic and focal encephalitis." If you have access to Nothnagel's System, issued in the late 90's, it would be well to read over an article of Leichtenstein's on the subject of encephalitis following influenza; cases which surely belong to precisely the same category as those now under discussion are there presented. Personally, I am of the opinion that although the germ of encephalitis lethargica has not been discovered, it is nevertheless, a postinfluenzal affair.

The disease first appeared in Vienna, then in Germany, and in England early in 1918. True, not many cases occurred in our own country immediately during the sweep of the first spread of influenza, but it quickly developed after the first burst of the infection, and again this winter in association with the second outbreak of influenza the cases of encephalitis lethargica have appeared. Although no germ has thus far been demonstrated in these cases, I am of the opinion that when one is found it will be either the influenza bacillus of Pfeiffer, or a germ having symbiotic relations with the influenza bacillus. Certainly, the pathologic picture is one of infectious inflammatory disease, and therefore of bacterial origin.

Discussion of Symptomatology.—If one studies the literature of encephalitis lethargica one will find four types of cases. There are, first, the fulminant, or foudroyant, types, which are very malignant, fatal in a few days, and which exist under the picture of acute mania, without any lethargy whatever. These cases would be very difficult to diagnose if it were not for the fact that they are associated with other cases of encephalitis lethargica.

Contrasted with these is the second type, the mild or abortive cases, with or without localizing signs, which are also hard to diagnosticate except in the presence of other cases. They betray themselves frequently by the ocular palsies, the double vision, or some of the characteristic manifestations of the disease.

A third type consists of those in which there are general disturbances of function without localizing signs, which are not mild or abortive, but which run a course of from four to eight weeks, with fever and headache, asthenia, and lethargy.

Last there is the common or usual type, which is that with localizing signs. This type may be divided into several subdivisions. There are, first, those cases in which double vision is an early and prominent sign; cases in which involvement of the oculomotor nerves is the early, salient feature. These are of the type which might be called polioencephalitis anteriora acuta. Second, there is the type in which there is disturbance chiefly of the brain stem, pons, and medulla. These are cases in which the seventh and twelfth cranial nerves are predominantly affected. They belong to the types of cerebrobulbar and pontobulbar polioencephalitis. Third, there is the type of case in which the brunt of the disease seems to be exerted upon the cerebral cortex. These are the delirious and maniacal cases. Usually these cases are not purely cortical, but show some involvement of the central gray matter. These cortical cases are also betrayed by the choreiform movements which take place in some of the individuals, especially in the cases like the one which we are just presenting. Just as in chorea minor, the choreiform movements are really cortical in origin, so also in lethargic encephalitis the probabilities are that the choreic movements observed are caused by the pathology found in the cerebral cortex. Fourth, there are the types which suggest involvement of the spinal cord, chiefly of the pyramidal tracts, sometimes of the direct cerebellar and possibly at times of the columns of Goll and Burdach. Associated with these are the ataxic types, whether of cerebellar or spinal origin it is difficult to say. Last, there is the so-called polyneuritic type, or, as I would prefer to call it, the meningoradicular type. There is no true polyneuritis. Instead, the pains which are observed and which, as in this case, lead to the diagnosis of polyneuritis, are really due to involvement of the spinal roots. This patient might be said to show the polyneuritic type, but she really does not; she shows instead the meningoradicular. This is proved by the fact that the spinal fluid was under tension and that it showed 105 cells per cubic millimeter. This indicates an involvement of the meninges with exfoliation of cells, and it also suggests an involvement of the meninges about the posterior roots, which in this case were the roots of the brachial plexus.

The types of cases with localizing signs are usually mixed. For example, the same patient may show all of the localizing signs, or certain combinations of them.

Differential Diagnosis.—In the differential diagnosis of encephalitis lethargica, botulism must be considered. In botulism the symptomatology is practically that of acute bulbur palsy, and the cases are not unlike those of polioencephalitis posteriora acuta, or of combined bulbar polioencephalitis. There is, however, one marked difference between botulism and lethargic encephalitis. Botulism is sudden in origin, quickly affects the medullary centers, and leads to the well-known picture of bulbar palsy within a short time. It does not have an incubation stage of seven days, but develops very rapidly after the food containing the Bacillus botulini has been ingested. Moreover, it is practically a uniformly fatal disease, and in this way also differs from encephalitis lethargica. Furthermore, it has neither the asthenia nor lethargy in its symptomatology.

The Course Taken by Lethargic Encephalitis.—As a rule, diseases have certain definite characteristics. Infectious diseases usually travel along on their course with such an average degree of exactness that one can tell in a given case about how many days an infection has been in existence. This holds true in most cases of encephalitis lethargica. There is in practically all of the cases an incubation stage of seven to fourteen days. That was well shown in this patient. She was first taken ill on January 4th, on Sunday, but did not get sick enough to see a physician until the 11th, when she was at once sent to the hospital, and it was not until that night that her fever began. Some patients have a longer incubation stage than one week; in some instances the period has been extended to three weeks, but in the majority of cases seven to fourteen days seems to be the average.

The stage of incubation is followed by the acute stage, which lasts, as a rule, not much less than four weeks, and rarely more than three or four months. This is the stage of fever, of asthenia, and of lethargy. This is the stage of the various cranial nerve palsies, which are transitory, quickly clearing up. During this stage the patient at first may show a good deal of maniacal

delirium, especially at night, but after the second week, and beginning with the third, lethargy and asthenia become the dominant features, and persist at times weeks after the paralyses have cleared up. In a girl of seventeen years, in a man forty-two years of age, and in a married woman twenty-three years of age the average duration of the acute stage was four to five weeks. In one case of which I have knowledge the acute stage lasted two or three months.

Lastly, there is the stage of convalescence, which is very slow and of uncertain extent. It is usually not less than four weeks in duration, and may last two or three months. During this stage there is usually complete recovery, but not in all instances, for in some of the English cases certain permanent defects have remained. In general, the time the patient quits the bed marks almost the exact midpoint of the disease.

Treatment of Encephalitis Lethargica.—What is the treatment? At present this is problematic and largely empirical. Believing, as I do, that the disease shows a certain amount of meningoradicular and meningo-encephalitic involvement, it is clearly rational to propose that hexamethylenamin be given. This can be administered in 5-grain doses four or five times daily, the only precaution necessary being to prevent irritation of the urinary bladder. A certain amount, about 10 per cent., of the hexamethylenamin will be secreted in the cerebrospinal fluid and may there exert its antiseptic action. Another remedy which also has some reason for being used is sodium cacodylate. We know that this remedy has a beneficial effect in luetic meningo-encephalitis, and it is not foolish to believe that it might also have an effect in the meningo-encephalitis of lethargic encephalitis. We have accordingly made use of both of these remedies in the case of this patient. Indeed, we went a step further and gave her two injections of neo-arsphenamine at intervals of three days. Whether these injections in this particular instance were of curative value we do not know, since one case is not sufficient to base an opinion upon, but certainly the effect has not been untoward, and the improvement which the patient made, though it may have been entirely spontaneous, with some

reason, may be partially ascribed to the remedy given. Aside from hexamethylenamin, neo-arsphenamine, and sodium cacodylate, I know of nothing which I could propose for the treatment of this disease. We have nothing which can control the tendency toward infiltration of the vein walls with lymphocytes and polymorphonuclear cells, and we have nothing which can prevent the miliary hemorrhages. In the latter stages of the disease, after the subsidence of the fever, it is rational to propose that iodids be given, either in the form of iodalbin or of the old standby, iodid of potassium.

Prognosis of Encephalitis Lethargica.—What is the prognosis in cases of encephalitis lethargica? For the most part, in cases which have originated in the United States, the prognosis has been good. Four cases which I have seen have all recovered, and 4 are still living and are expected to recover. Of 12 cases which an associate of mine, Dr. Bassoe, has seen, 2 have died and 10 have recovered. In the English cases the mortality was not very high, not exceeding 20 per cent. The cases which have occurred in the United States have, on the whole, seemed to be milder than those which have occurred abroad. As a rule, though severe cases may result fatally, we look for complete recovery or, rarely, for recovery with residual defects. Those cases which die usually die early from the toxemia or from the multiplicity of miliary hemorrhages in the gray matter. The patient whom you have just seen might have died if the disturbance of her cardio-inhibitory center had been greater, more prolonged, and if there had been disturbances also of her respiratory center. As it was, there was a time when we hardly dared hope for her recovery, especially in view of the extremely rapid pulse, high temperature, and mania. Her worst night was probably the night of January 11th to 12th, at the time of the sudden development of her fever. Her most critical period was during the paralysis of the cardiac inhibition, when her pulse remained at 150.

MEDIASTINAL TUMORS

Full Discussion of Mediastinal Tumors, Both Benign and Malignant, with Classification; Symptomatology; Autopsy.

History of the Case.—The patient, a Polish laborer, forty-three years of age, comes to the hospital complaining chiefly of dyspnea and cough. He states that his present trouble began about two years ago; that about nine months ago he went to the Cook County Hospital, where he remained for a time under medical treatment. He made a partial recovery, so that he was able to resume work, but in a comparatively short time grew worse again and has been compelled to give up his job. His wife recently died in a sanitarium, of pulmonary tuberculosis. He has two children who are living and well. There is no history of miscarriages. He gives a somewhat doubtful history of a Neisserian infection, but declares that he never had a chancre.

Besides the dyspnea he complains of cough which at times keeps him awake during the night. The cough is productive and the sputum is streaked with blood. He has no difficulty in swallowing food and there is no hoarseness. He does not complain of headache, vertigo, or of edema of the extremities. The symptom which has most attracted his attention is the great dilatation of the veins over the anterior surface of his trunk. These enlarged veins were not in existence nine months ago when he went to the Cook County Hospital, but have become quite prominent in the last few weeks. The probabilities are, from his history, that these venous enlargements date back about eight months.

On entrance the patient's temperature was 97° F., his pulse 104, and his respirations 28. A leukocyte count made at once showed 8400 cells per cubic millimeter. The blood Wassermann reaction was negative. The urinalysis showed a specific gravity of 1027, acid reaction, no albumin, no sugar, no casts, an occasional white cell, no red cells, and no epithelial cells. The

examination of his sputum made on entrance showed no tubercle bacilli, a few pneumococci, a few staphylococci, and the Micrococcus tetragenus. The sputum contained a large number of pus-cells and quite a number of red blood-cells, even when it did not appear red streaked.

The first night he was in the hospital he slept for about six hours, but was prevented from having a good rest by reason of the cough. On the fifth day after his entrance he showed a temperature of 99° F. for the first time. On the sixth day his temperature reached 99.8° F., and at the end of the first week in the hospital his maximum temperature was 101° F. His blood-pressure, taken after he had been in the hospital for five days, showed a systolic pressure of 100 and a diastolic pressure of 40. After his temperature first began to rise it never again fell to normal.

Physical examination showed as the most prominent feature a dilatation of the superficial veins of the abdominal wall and chest. It will be noticed that there is a very marked dilatation of both the superficial epigastric and superficial circumflex iliac veins, the right superficial circumflex iliac being larger than the left. Note the enormous size of the superficial epigastric veins. Each one is nearly $\frac{1}{4}$ inch in diameter. Note also the dilatation of the veins above the umbilicus. These veins form an anastomosis between the superficial epigastric and superficial circumflex iliac, on the one hand, and the external mammary on the other. Note the large size of the blood-vessels in the upper right part of the thorax (Figs. 250, 251).

The next most prominent feature in regard to the patient is the dyspnea. You will notice that he breathes with more or less difficulty and that his respiratory rate is approximately 30. You will also notice, if you examine closely, that the expansion of the right lower thorax is less than the expansion of the corresponding portion of the left chest. You will also note that there is a very slight permanent bulging of the lower right thorax as compared with the lower left. On percussion there is a marked dulness with a sense of resistance in the lower right chest, especially in the back, close to the spinal column. This

dulness extends from the seventh rib downward and is lost in the liver dulness. You will notice that on the left side the resonance is much more normal than the corresponding area.

There are certain important negative signs. There is no pupillary inequality, no tracheal tug. There is no ascites and no edema of the legs. The liver is slightly displaced downward.

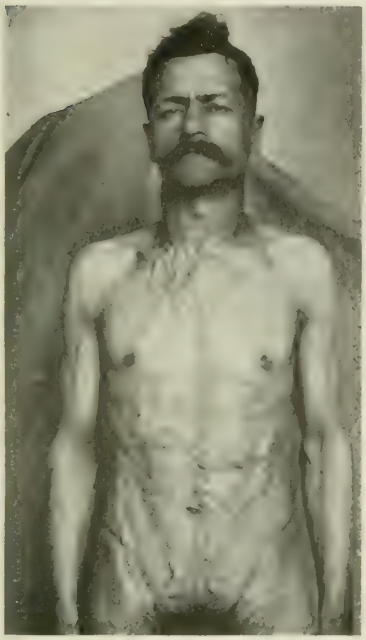

Fig. 250.—Dilated superficial abdominal veins in case of posterior mediastinal tumor due to compression on the vena cava inferior below the opening of the hepatic vein.

Fig. 251.—Dilated superficial abdominal veins in case of posterior mediastinal tumor due to compression on the vena cava inferior below the opening of the hepatic vein.

The spleen cannot be felt. The heart is displaced somewhat downward and backward and to the left. You will also notice that the man's color is not good, that he is slightly cyanotic. This cyanosis may be an ordinary respiratory cyanosis, but it is probable that it is somewhat increased by venous pressure. You will recall that his blood-pressure was 100 systolic and 40 diastolic.

This is an important fact, because it may mean that something interferes with the normal flow of blood into the ventricles of the heart. For example, if a mediastinal tumor presses upon the heart sufficiently it may make it impossible for complete ventricular diastole to take place. The result is a small and rapid pulse, with a low systolic and diastolic pressure, exactly such as we find in this patient.

The diagnosis in this case is extremely simple. There is no condition which can produce such a great distention of the superficial veins except one, and that is compresssion upon the vena cava inferior. If the pressure upon the vena cava takes place *above* the point where the hepatic vein enters, there will be both dilatation of the superficial veins and an ascites because of the portal blockade; whereas if the pressure be exerted upon the vena cava inferior *below* the point where it receives the hepatic vein, then there will be no interference with the passage of portal blood between the liver and the heart. The only interference will be in the passage of blood from the legs and internal and external iliac veins and their tributaries. It will be necessary if there is a blockade in the lower thorax for the blood which should ascend in the vena cava to pass downward through the common iliac, internal and external iliac veins, thence down the common femorals and up over the surface of the abdomen through the superficial circumflex iliac veins, or from the external iliac veins up through the superficial epigastric veins. The blood thus ascending in this manner can make its way through the external and internal mammary veins to the subclavians, through the venæ innominatæ to the vena cava superior, and so to the heart. It is obvious that such a round-about collateral venous circulation must lead to a good deal of embarrassment of circulation and accentuate the cyanosis.

Since only pressure upon the vena cava below the point of entrance of the hepatic vein can cause the condition which this patient presents, it is obvious that the diagnosis must account for such pressure. Only two things can exert such pressure, and these are mediastinal tumor and aneurysm. How can these two conditions be differentiated?

By physical examination it is very difficult to make such a differentiation, because there is not a single sign found in aneurysm which may not be found in mediastinal tumor. In other words, there is not a single differential point. Even tracheal tug has been described in mediastinal tumor and is often absent in aneurysm. The only safe method of differentiating is by means of the *x*-ray. This patient was skiagraphed the day after his entrance, and I will show you the *x*-ray plate. You will notice the large shadow situated in the lower mediastinum and spreading to the right. You will notice that the heart is displaced somewhat to the left. The shadow of the aortic arch is not noticeably enlarged. The left lung is quite transparent and shows a normal apex and very little peribronchial streaming. The right lung is reduced in bulk at least one-third in comparison with the left, and shows more infiltration in the region of the right hilum than is to be found in the left lung. Such a picture leads to the presumption of a neoplastic mass in the posterior mediastinum.

Mediastinal tumors in general are classified as benign and malignant, but any benign tumor may, by reason of the pressure which it exerts, be fatal. Benign mediastinal tumors are to malignant tumors in the ratio of about 1 to 10. The ordinary benign tumors are dermoid cysts, fibromata, lipomata, enchondromata, and enlarged lymph-nodes. The malignant tumors are sarcomata and carcinomata. Carcinomata occur about three times as frequently as sarcomata, according to later statistics. Formerly sarcomata were said to be more numerous. As a rule, such malignant tumors are primary; the ratio between primary and secondary mediastinal tumors being about as 8 to 1.

The malignant tumors, as a rule, spring from the thymus and its remnants, sometimes from the thyroid, but more frequently from the connective tissue and the lymphatics. There is some slight diagnostic value in the localization of the mediastinal tumor. For example, tumors of the anterior and superior mediastinum are far more apt to be sarcomata than are those of the posterior mediastinum, which are usually carcinomata. The metastatic mediastinal tumors are usually secondary to car-

cinomata of the breast and lung, but occasionally they are derived from primary tumors in the bones, gall-bladder, and stomach. In general, the sarcomata and carcinomata not only compress veins, but they actually grow into them, thrombosing and plugging them.

The symptomatology of mediastinal tumor naturally depends upon its location. If, as in this case, the mediastinal tumor is in the posterior mediastinum, it will produce few pressure signs until it has reached a very large size. On the other hand, if the tumor is in the anterior mediastinum, it may produce marked pressure symptoms early by reason of its effect upon the sternum and ribs, its compression of the blood-vessels, trachea, bronchi, recurrent laryngeal, and sympathetic nerves. In tumors of the posterior mediastinum the only physical signs which one will find will be, as in this case, possibly a slight bulging of the posexterior or lateral chest wall with limitation of the respiratory cursion, dulness, and an increased sense of resistance, slight suppression of the respiratory sounds, with a fairly good conductivity of the cardiac sounds, the presence of marked dyspnea, cough—usually unproductive, but occasionally productive in the later stages—and usually fever, with loss of weight and of strength. These are the signs which we have in this case, and with the *x*-ray findings they enable us to make a diagnosis of posterior mediastinal tumor.

Inasmuch as such tumors are mostly carcinomata, it is a fair inference to suppose that this will be the type of tumor found in this individual at autopsy.

NOTE.—This patient died on the twenty-third day after his admission to the hospital. Toward the end his temperature ranged from 99° to 101° F., and his breathing became extremely difficult. Suddenly, on the last night of his life, his pulse rose from 80 at 6 P. M. to 110 at 9 and 140 at 10 P. M. His respiratory rate, which had been about 32 to 34, increased to 46. The last temperature taken showed 101.4° F. (axilla) at 9 P. M. He died at 11 P. M. An autopsy was secured, and examination revealed carcinoma of the posterior mediastinum.

CLINIC OF DR. ARTHUR F. BYFIELD

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AN ANALYSIS OF THE MORE IMPORTANT CAUSES OF ERRORS IN DIAGNOSIS

IT is the imperative *duty* of the teacher of medicine, in the course of his clinical work, to impress upon his students, among other things, these two fundamental ideas: (1) By no means every case, even after a prolonged and thorough study, is susceptible of diagnosis during the lifetime of the patient; (2) in a great many cases the failure to establish a diagnosis—we imply a *correct* diagnosis to the extent of the limitations imposed even by our modern methods of analysis—is due to factors which can, to a great degree, be eliminated.

With reference to point one: Many a student is graduated with the idea that a correct and absolute diagnosis can be arrived at in *every* case. Some teachers believe that the student can appreciate only the type case, and laboring under this idea they permit each patient to leave the clinic *finally and positively diagnosed.* It is with regret that we must add that instructors of this type are sometimes guilty of failing to mention certain data which throw doubt upon the correctness of their diagnoses. They are not necessarily dishonest in their methods, but are obsessed with the idea that the student must not be allowed to wander from the straight and narrow path of medical orthodoxy.

Rigidity in diagnosis may be fostered in another way, corollary to that already mentioned: a probable diagnosis is made during one clinical hour; this is allowed to stand as the correct diagnosis because at subsequent hours the data of the further

study of the case are not mentioned or the results of operation or autopsy are not brought forward, especially if these have shown the incorrectness of the preliminary diagnosis.

Such methods of study are no longer defensible, at least in our schools of the higher type. The teacher must emphasize—and emphasize frequently—that this case and that case allows only a probable diagnosis. In some instances positive *intra vitam* diagnoses can never be reached, and the student has the right to know this; of this type, a part go on to recovery without an unimpeachable diagnosis, while the remainder are solved only by exploratory operation or at autopsy. Nor are the cases few in which neither the surgeon nor the pathologist is able to find a satisfactory explanation of the clinical picture. In other instances the further study of the condition will enable the physician to reach a correct conclusion.

We do not mean to take the position that the teacher should confuse the student with unnecessary diagnostic detail or give him the impresssion that the typical does not form the groundwork of diagnosis. But he must not be allowed to leave medical school with an inflexible, one-track mind which cannot countenance objection to the cocksureness of his diagnosis. He must not have that unfortunate mental twist which allowed a certain teacher to say "Once, I almost made a mistake."

The foregoing is by way of introduction to our second point—the more important causes of the failure to establish a diagnosis in certain cases. Some cases, we repeat, are not susceptible of diagnosis however carefully they are studied; others—and these are the ones forming the theme of this clinic—offer no great obstacle to a solution if certain, very obvious, faults are eliminated.

The common causes of errors in diagnosis may be tabulated thus:

1. Incomplete or incorrect case-history taking.
2. Incomplete examination of the patient.
3. Ignorance of certain pathologic complexes. This is not the ignorance of poor schooling, but of conditions which our texts fail to emphasize sufficiently as clinical complexes.

4. Failure to explain the atypical in a case, or, conversely, failure to build up a diagnosis upon the typical.

5. A plus of laboratory detail and of ultramodern methods at the expense of sound judgment and good sense.

6. The riding of a hobby.

We shall attempt to analyze and illustrate these selected causes of diagnostic error:

1. **Incomplete or Incorrect Case-history Taking.**—The importance of this phase of the diagnostic routine can surely not be overemphasized. As we have often stated, a ragged anamnesis may lead the diagnosis into an absolutely wrong channel, while, on the contrary, a complete, well-rounded, well-analyzed case-history may practically establish the diagnosis.

We shall limit our discussion of case-histories at this time to two faults which appear especially flagrant. The first is that of the *incomplete history*. This is as likely a source of diagnostic error as is the incomplete examination of the patient. An incomplete history is likely to arise from two causes: first, from the use of printed forms, no type of which can be applicable to *every* case, and is bound, therefore, to limit at one point or another the information to be obtained from the *particular* case; and second, from the mere failure to question the patient concerning the data falling under the several accepted subheads of the history.

Illustrations of the inadequacy of the printed forms for the case-history will occur readily to every one who is in the habit of writing histories. For example, a small space devoted to the patient's complaint will discourage the very essential analysis of the complaint, *i. e.*, mode of onset, development of the symptoms, status of symptoms when the patient presents himself to the physician. Or the list of previous illnesses which happen to be printed on the card will often lead to a failure to ask concerning diseases—*or symptoms*—which do not happen to be thus printed. Cough, headache, jaundice, edema, even tonsillitis, rheumatism, and malaria may not be specifically asked about, because the physician has fallen into the groove initiated by the particular

case-history form he employs, and that particular form does not mention these symptoms or conditions.

Equally dangerous and misleading is the incomplete history due to haste or carelessness on the part of the physician. The latter may fail to bring out the fact that there have been in the past attacks of abdominal pain, which, when properly analyzed, indicate that the present—perhaps ambiguous—abdominal condition is renal colic, or pyelitis secondary to renal stone, or cholecystitis on the basis of a pre-existing stone, or appendicitis, or a perforation of a gastric or duodenal ulcer. We are all familiar with the fact that many, if not most, cases of so-called "indigestion" are reflex, proceeding from lesions elsewhere, and that often the previous history alone will enable one to localize the organic source of the reflex.

We shall make no attempt to exhaust this matter of the incompleteness of the anamnesis in the failure to analyze exhaustively the *present complaint* of the patient (to which we shall call attention directly); in the neglect to ferret out *past illnesses* which may have a bearing upon the present complaint; in the slurring over of the patient's *daily routine* as to work, sleep, diet, use of alcohol, tobacco, drugs, etc.; in the matter of the *family history*, particularly as to tuberculosis, carcinoma, diseases of the nervous system, and possibly cardiorenal processes; and in the details of the other subdivisions of the history (venereal, menstrual, etc.).

The second glaring fault of the average case-history concerns the matter of a thorough analysis of the patient's symptoms. This fault, though especially likely to creep in in connection with the so-called present illness of the patient, may affect to a considerable degree every part of the anamnesis. (Indirectly, this has been brought out above relative to previous sickness having a bearing upon present illness.) The fault consists partly in the accepting of the patient's statements as to symptoms of diseases at their face value and partly in the failure to obtain all the information possible about a given symptom or process. As illustrations of the former may be cited headache, pain in the "stomach," rheumatism, sciatica, hernia, and shortness of breath,

which when fully analyzed prove to be, or to have been, respectively, pain in the posterior cervical region, fulness in the epigastrium at a variable time after meals, flat-foot, the lightning-pains of tabes, varicocele, and inability to draw a full breath.

The second point—the matter of obtaining all the information possible about a given symptom—is not so likely to lead to diagnostic error (it is assumed that the symptom has been proved to be as stated by the patient) as it is to limit the value of the history. We are very fond of using the symptom *pain in the stomach* to illustrate this point. Actual pain in the gastric region, with no further qualifications, may point to a number of conditions; the same symptom, exhaustively analyzed, as to character, occurrence (periodicity, relation to meal time and to the character of the food), association with hematemesis, radiation, relief by sodium bicarbonate or vomiting, association with constipation or distention, etc., may suggest with a high degree of probability, even before the actual examination of the case has been begun, peptic ulcer, the gastric crises of tabes, abdominal angina, or other conditions.

Similarly, each and every symptom, past and present, and the details of previous disease must be fully analyzed and looked at from every point of view to enable the physician really to get valuable material from the anamnesis. Analysis of this sort plus a complete history in every case, unhampered by a set formula for questioning the patient, will do away with a considerable proportion of wrong diagnoses.

2. **Incomplete Examination of the Patient.**—The necessity of emphasizing the part played by the inadequate examination—both physical and laboratory—as a cause of incorrect diagnoses would seem to be a matter of the dark ages of medicine rather than of the present time, yet, it is a very common fault and one not entirely confined to the physician of poor training. As we have said in another place, the *average* physician who takes a careful case history and who makes a thorough examination of his patients will have to his credit a larger number of correct diagnoses than will his more brilliant colleague who is satisfied with slipshod methods.

That the incomplete examination is an important factor in the theme we are developing is well shown by certain data which we shall quote from a paper read by James S. Ford at the eleventh annual meeting (1915) of the National Association for the Study and Prevention of Tuberculosis. In this paper Ford analyzes 1000 cases admitted to the Gaylord Farm Sanatorium, as follows:

(1) The 31 non-tuberculous cases of the series consulted 56 different physicians, of whom:

(*a*) Thirty-seven (66 per cent.) made a physical examination, only one of these in addition making an *x*-ray study;

(*b*) Six (10.7 per cent.) examined the chest and the sputum;

(*c*) Six (10.7 per cent.) examined the chest, took the temperature, but made no sputum examination;

(*d*) Four (7.1 per cent.) examined the chest, took the temperature, and examined the sputum;

(*e*) Three (5.3 per cent.) made no examination whatever.

(2) The 148 incipient cases consulted 254 physicians, of whom 160 (62.9 per cent.) made an examination of the chest only, and 11 of these made this examination through the patient's clothing. As for the remainder:

(*a*) Two (0.8 per cent.) took the temperature only;

(*b*) Three (1.1 per cent.) examined the sputum only;

(*c*) Twenty-seven (10.7 per cent.) examined the chest and sputum, but did not take the temperature;

(*d*) Fifteen (6 per cent.) examined the chest, took the temperature, but did not examine the sputum;

(*e*) One (0.4 per cent.) took the temperature and examined the sputum, but made no physical examination;

(*f*) Seven (2.8 per cent.) examined the chest, took the temperature, and examined the sputum;

(*g*) Thirty-nine (15.3 per cent.) made no examination whatever, but treated the patients for various ailments, such as "run down," anemia, malaria, and "walking typhoid."

"Here we have only 3 per cent. of the doctors making use of most of the diagnostic methods available, while over five times that number made absolutely no effort to find the seat of the trouble, but, by their negligence or carelessness, allowed a case

with an excellent opportunity for arrest and a return to normal life to go on to a far-advanced stage."

Ford's figures relative to the moderately advanced and advanced cases are just as startling in the matter of errors of omission as are those just quoted, but it will avail nothing to burden you with them. We shall, however, give you the results of the several means of diagnosis made use of in the 1000 cases as a whole. The 1000 cases consulted 1940 physicians, of whom 1085 (55.1 per cent.) made a physical examination only, and of this number, 151 did not deem it worth while to have the patient strip; 13 (0.7 per cent.) took the temperature only; 14 (0.7 per cent.) made a sputum examination only; 381 (20 per cent.) made a chest and sputum examination; 114 (6 per cent.) examined the chest, took the temperature, but made no sputum examination; 3 (0.02 per cent.) took the temperature and made a sputum examination, but made no physical examination; 133 (7 per cent.) made a chest examination, took the temperature, and examined the sputum; 197 (10.2 per cent.) made no examination of any kind.

Comment on the foregoing figures is scarcely necessary. The contrast of 133 out of 1940 physicians (7 per cent.) making use of simply the fundamentals in the diagnosis of pulmonary tuberculosis, as against 197 men (10.2 per cent.) who made no examination whatever is so striking as to be almost unbelievable.

This is not a clinic on tuberculosis, and the data quoted are made use of simply because they are available, whereas there is little of a similar nature in relation to other diseases. To a very great extent, however, we believe that a lack of thoroughness in examination applies to morbid processes in general. Diagnoses cannot be established by the examination of the patient's tongue alone, as many patients seem to think from past experiences. Every case, whatever its nature, demands a *complete, routine examination* following the lines of the generally accepted definition of this term; and symptoms referable to special organs require particular study, physical and chemical, so far as the equipment of the general practitioner or available consultation permits. The errors of omission cited in connection with pulmo-

nary tuberculosis are no less excusable than are the failure in the routine examination to test the pupillary reactions, to examine the knee-jerks, to make a urinalysis, to determine the blood-pressure, to examine the heart, lungs, and abdomen by the usual methods of physical diagnosis (inspection, palpation, percussion, and auscultation), etc. And to attempt to treat any condition without insisting upon the patient's removing the necessary clothing is nothing short of malpractice.

In recent years there has been a tendency on the part of some physicians to go to the other extreme in the matter of examinations, in that they have allowed the results of ultradetailed studies, chiefly on the laboratory side, and often not of entirely ambiguous interpretation, to cloud the obvious in the particular case. This aspect of diagnostic error will be considered in another place.

3. The third source of diagnostic error we have called **the ignorance of certain pathologic complexes.** To a certain degree this fault can be laid at the door of our medical schools; furthermore, it is not an entirely avoidable fault. More particularly, it is a fault due to the text-book method of teaching, especially by men who have not the experience to interpret and to interpolate. A text-book in any branch of medicine can scarcely be complete enough to dwell with any degree of emphasis upon the complex, which is more properly the subject of the special monograph or paper; nor is the medical curriculum long enough or elastic enough to permit of much wandering from the well-worn paths. The small bedside clinic, teaching from case records and special courses, will tend more and more to obviate the fault we are about to illustrate.

(*a*) *Chlorosis with a Normal Blood-picture.*—The majority of students leave medical school with the impression that the hematologic criterion of a true chlorosis consists of a low color index, *i. e.*, a disproportionate reduction of the hemoglobin as contrasted with the red blood-cells. In the great majority of cases this is, indeed, true, but in the occasional case the hemoglobin and red cells are practically normal, and yet there is present in well-developed form the chlorotic clinical picture (pallor,

with the characteristic turgor of the face, sleepiness, lack of energy, dyspnea on exertion, headache, systolic murmurs, venous hum, menstrual disturbances, etc.). Iron is as truly a specific in such cases as it is in those with well-marked blood changes. These variations from the type are strongly indicative of the truth of the hypothesis that the fundamental change in chlorosis is not in the blood-forming organs, but elsewhere (ovaries, internal secretions as a whole).

(*b*) *Cirrhosis of the Liver.*—The average text-book concerns itself with the type case of the Laennec or of the Hanot forms. The extremely common occurrence of the so-called *mixed types* is generally not mentioned. Clinically, these mixed forms do not fall readily into the category either of the Laennec or Hanot cirrhoses; pathologically, changes common to both are found.

We encounter, for example, cases with an alcoholic history (Laennec) which show periodic elevations of temperature (Hanot); slight or no enlargement of the liver, with the sharp edge of the atrophic organ (Laennec, though the alcoholic liver may, of course, be enlarged and the edge fairly rounded if fatty changes are considerable); jaundice (Hanot); enormous spleens (Hanot).

Such cases indicate that the cirrhoses in general are manifestations of a toxemia of variable kind and origin, which, depending upon factors unknown, produces type changes in one instance and blurred types in another. As we have pointed out on other occasions, the so-called Banti complex can readily be included in the category of the cirrhoses as an instance of the special affinity for the spleen of the hypothetic toxic agent.

(*c*) *Wide-spread Dilatation of the Bronchial Tree.*—To the average physician the term "bronchiectasis" suggests the saccular type, with its mouthful expectoration, signs of cavities, clubbed fingers, etc. Postmortem examinations revealing the frequency of multiple small bronchial dilatations or a general widening of the bronchial system demonstrate the inadequacy of this conception. On the clinical side, we are confronted often with the individual who shows, generally, a variable degree of pulmonary emphysema, who has periodic attacks of bronchitis

with profuse expectoration, often with elevations of temperature, with no demonstrable signs of cavity, no mouthful expectoration, no clubbing of the fingers, and roentgenologically either no conclusive evidence of bronchial dilatation or the picture of multiple bronchiectases (also, in both instances, as a rule, the *x*-ray signs of emphysema and chronic bronchitis).

Indeed, we have no doubt that this form of bronchiectasis is of far commoner occurrence than is that of the single large cavity.

(*d*) *Pyelitis*, with irregularly recurring pain, fever—often with chills or chilliness and sweating—due to the kinking of the ureter, with subsequently infected urine, of a low-lying kidney. We have seen several instances of this condition in the past few months. One case was diagnosed and treated as malaria, the misplaced kidney having been called the spleen; the other was regarded as a tuberculous pleurisy or a pulmonary tuberculosis. In both cases the diagnosis was established to our satisfaction, at least, by thorough urologic studies and by the results of treatment (padded corsets, forced feeding, etc.).

(*e*) *Epigastric hernia* as a cause of gastric disturbances. We know that the views as to the pathologic significance of this condition are diametrically opposed. It has been our experience that operation has caused a complete disappearance in a very great majority of the cases in which we have advised surgery. This condition is noted in this connection because, outside of the special article, there has been little mention made of the well-marked associated clinical complex—pain in the epigastrium, at a variable time after meals, vomiting in some cases, flatulence, heart-burn, and local tenderness.

(*f*) *Gastric Symptoms of Reflex Origin.*—The general texts are just beginning to emphasize the fact that the majority of cases of "indigestion" have their origin outside of the stomach. We have already mentioned epigastric hernia as an instance in point. Gall-bladder disease, chronic appendicitis, cardiac pathology, focal infection, pelvic disease, tuberculous spondylitis—in fact, disease in any organ—is likely to initiate gastric symptoms. Most functional gastric disorders are, we believe, of reflex nature, and an unlimited number of wrong diagnoses can

be avoided if, after first eliminating organic gastric disease, the source of the "indigestion" be sought outside the stomach, and not by treating such cases symptomically.

Illustrations of this phase of our topic can be multiplied indefinitely, but we believe we have given a sufficient number to make clear our point, viz., that the text-book of medicine can only be a reference book; it cannot portray the amazing interweaving of morbid processes, the pictures within pictures, as they are presented to us at the bedside.

4. **A lack of insistence upon the typical** constitutes our fourth source of diagnostic error. It may appear that we are reversing the opinions set forth in the preceding paragraphs, *i. e.*, as to the necessity of recognizing clinical pictures not included in the text-book types. This is only an apparent contradiction. We reason from the standpoint of the typical, and it is only when we discover facts out of keeping with the type that we must turn to the anomalous.

The point we shall try to make in this fourth subhead of our theme is this: We are not, as a rule, justified in making a diagnosis, even though all or nearly all of the clinical and laboratory data support this diagnosis, if we are confronted by an anomalous symptom of *constant* character, or by the absence of an expected (important) symptom. This statement, of course, demands certain qualifications. Thus, the diagnosis loses nothing in strength if the presence of the inharmonious or the absence of the expected symptom can be satisfactorily explained. Again, whatever weak links there may be in the diagnostic chain, findings of a certain character are definitively diagnostic (typhoid bacilli in the blood, tubercle bacilli in the sputum, malarial parasites in the blood, tubercle bacilli in the cerebrospinal fluid, Treponema pallida in an ulcer, etc.).

Illustrations of our point are plentiful:

(*a*) *The persistent absence of both bacillemia and the Widal reaction* in cases otherwise pursuing the course of typhoid fever. We recall many cases falling into this category in which the constant absence of these symptoms was not given its proper weight. (One naturally must not lose sight of the fact that a

bacillemia is most likely to be present during the early days of typhoid fever and the Widal reaction from the tenth to fourteenth days on.) These cases have a typhoidal onset, gradual, with malaise, anorexia, fever, headache, cough, weakness, and perhaps abdominal symptoms; the temperature is characteristically typhoidal; the pulse is relatively slow and sometimes dicrotic; the tongue is dry and heavily coated; a leukopenia is frequently present or, at any rate, there is no increase in the white blood-corpuscles; the abdomen is often somewhat distended and sensitive to pressure; the spleen is generally enlarged, and an exanthem is frequent. The latter may have all of the characteristics of the rose-spot, or it may be more diffuse and persistent than the rose-spot, or be hemorrhagic in character, in which events it may readily be regarded as an atypical typhoid exanthem.

In several of these cases we were put upon the right track by the persistence of the headache, and especially by the increase in its severity, and subsequently by the appearance of neck rigidity and other manifestations of meningitis. A spinal puncture revealing, first, the findings of an inflammatory meningeal condition, and second, the tuberculous character of that inflammation (tubercle bacilli) explained the absence of typhoid bacilli in the blood and of the Widal reaction. And yet, we recall a case conforming in all particulars to what we have just described, with the exception of the signs of meningeal mischief, which went to autopsy with the diagnosis of typhoid fever and proved to be tuberculous meningitis.

While speaking of typhoid fever we may call attention also to the presence or absence of certain findings which should not be allowed to go unexplained before the diagnostic routine is completed. A leukocytosis, the absence of a splenic tumor (assuming that tympanites does not prevent conclusions as to the same), a fever which drops to normal from time to time (during what should be the height of the disease, and without cause), a fairly clean, moist tongue, a good appetite, a feeling of well-being or an inclination to be talkative—these and many other variants from the usual picture should be viewed with suspicion in a case otherwise pointing to typhoid fever.

Besides tuberculous meningitis and other forms of miliary tuberculosis our experience has been that cases with persistently negative blood-cultures and Widal reactions have ultimately proved oftenest to be sepsis (septicopyemia, ulcerative endocarditis, etc.), malaria, Brill's disease, and tuberculosis.

(*b*) *Low-color index, constantly large numbers of nucleated red blood-corpuscles, absence of leukopenia, etc.*, in cases otherwise suggesting pernicious anemia.

As a rule *one* of these symptoms is present in a case, likely, on the basis of the remaining findings, to be called pernicious anemia. (We refer in this discussion only to the idiopathic, Addisonian type of pernicious anemia.) It is true that under certain conditions—usually a period of marked regeneration—the color index may be somewhat less than 1, that the circulating blood may be literally flooded with erythroblasts, and that there may be a moderate leukocytosis. However, this is both exceptional and, if present, transitory. A low color index generally means an anemia of the secondary type; *constantly* large numbers of nucleated red-cells point rather to some such condition as metastases into the bone-marrow from a malignant growth or an acute leukemia, and the absence of a leukopenia to an anemia of discoverable etiology.

We have no intention of entering into the discussion as to whether pernicious anemias should be subdivided into those with and those without discoverable cause; we insist merely upon the point that if one is of the opinion that that term "pernicious anemia" should be employed only for cases of apparent idiopathic nature, the presence of an unexplained atypical feature such as those mentioned should suggest the possibility of other pathology (carcinoma, especially of the stomach, infectious endocarditis, chronic nephritis, cirrhosis of the liver, lead-poisoning, etc.).

Not infrequently, as we have pointed out elsewhere, one encounters blood-pictures characteristic of pernicious anemia in all particulars except for the presence of a fairly high eosinophilia. It will be recalled that, as a rule, the eosinophils in this condition are sometimes normal in number, generally decreased,

and often absent. An increase is rare, and usually means that the case is not one of pernicious anemia. Even in those forms of pernicious anemia due to the presence of an intestinal parasite (Bothriocephalus latus, in particular) as the anemia becomes of high grade the eosinophils gradually disappear from the peripheral blood. Therefore the general rule may be formulated: A severe anemia with an associated eosinophilia is likely to prove to be of the so-called secondary type.

(*c*) The *diagnosis of malaria* in a case showing paroxysms of chill, fever, and sweat, *without the finding of the parasite*, is generally open to question. This is a very common diagnostic fault. There may be some excuse for the fault if the patient has had malaria or if he has been living in a malarial neighborhood. In cases of long-standing infection it may be difficult to find the parasite—especially that of estivo-autumnal fever—in the peripheral blood. Occasionally provocative doses of quinin or, as recently suggested, of strychnin may drive the parasite from the viscera; and sometimes, in the chronic cases, a diagnosis is perhaps warranted on the basis of the presence of a large amount of pigment in the blood, especially if it is in the leukocytes; and, lastly, the effects of treatment are of vast diagnostic worth.

If all of these criteria and diagnostic reinforcements fall by the wayside, the case can hardly longer be considered one of malaria. (We are assuming that a careful general examination has already excluded the more obvious causes of the febrile manifestations.) A masked septic infection is then to be sought (infective endocarditis, pyelitis, tonsillar infection, sinus infection, gall-bladder infection), or a tuberculous process. Though these last-mentioned conditions are frequently associated with a leukocytosis of variable degree, they may show either no change in the white cell count or even a slight leukopenia, thus conforming further to the malarial picture. And we must not overlook the fact that occasionally, even in malarial fever, particularly during the chill, a well-marked leukocytosis may be observed, while examination a few hours later will reveal a leukopenia.

(*d*) In a field so poorly defined as that of the *acute arthritides*

one must be especially conservative in the diagnosis of true rheumatic fever. A given case must closely approach the typical before a definitive conclusion is warranted. Localization in one or more large joints, simultaneous—not progressive—involvement of joints, absence of acid sweats, and especially a failure of the symptoms to yield to salicylates, properly given, are grounds for a possible revision of the clinical data. Such a review will generally reveal that certain data have not been fully gathered (particularly those falling in the category of previous illness) and that other data have not been sufficiently analyzed and properly interpreted. With the added information the diagnostic pendulum is likely to swing toward a septic arthritis, a gonorrheal arthritis, or the secondary arthritis of focal infection.

The foregoing examples of a lack of insistence upon the typical have been mentioned because we have recently been confronted with similar situations. Every clinical condition presents them in one way or another. In croupous pneumonia a slow onset, or the absence of herpes or of rusty sputum; in suspected scarlatina, the absence of a well-marked leukocytosis; the failure, after repeated examinations, to find tubercle bacilli in a pulmonary condition thought to be tuberculosis; a negative Wassermann reaction in a mediastinal condition called aneurysm; carcinoma below the usual age; "unresolved" pneumonia with a septic temperature curve—these are a few additional variations from the type demanding explanation before one can say the diagnosis has been established. And a failure to regard such variations lays one open constantly to diagnostic error.

5. **A plus of laboratory detail and of ultramodern methods at the expense of good judgment** is a fifth rather frequent source of diagnostic error. It constitutes the only ground for criticism of our modern ultrarefined methods of studying the patient, in that, in the hands of one whose clinical judgment is not ripe, it tends to make the detail loom too large in the picture as a whole.

Naturally, it is not the routine but the special laboratory

method which is likely to lead one astray. The point presents itself in several ways. In the first place, a *laboratory result may not be properly interpreted.* Take the von Pirquet test, for example. Scarcely a week goes by during which we do not encounter a case—in an adult—in whom the diagnosis of tuberculosis has been made on the basis of a positive von Pirquet reaction. The vast majority of such conclusions are wrong; first, because, as a general thing, a positive reaction in an adult means nothing, and further, granting, as some assert, that the degree of the reaction is of diagnostic significance even in an adult, no attempt has been made to interpret the positive reaction from the standpoint of its intensity. Meanwhile, an early hyperthyroidism, a low-grade infectious endocarditis, or an early malignancy is probably being overlooked.

As another example of interpretative error—in this case, of a routine test—may be mentioned the matter of a leukocytosis. Ordinarily the increase in the white blood-corpuscles is decisive enough to leave no room for doubt as to whether a leukocytosis is actually present. Not infrequently, however, the total count may lie, let us say, between 8000 and 10,000, and in cases in which a leukocytosis, even slight, is of significance (mild, recurrent appendicitis, chronic, infective endocarditis, chronic perforative gastric, or duodenal ulcer) a correct interpretation of the laboratory data is essential. We cannot attempt to set down a rule as to the upper limit of the normal white cell count, but our feeling is that counts above 10,000 are usually pathologic and that counts of 8000 to 10,000 are very frequently indicative of infection.

The foregoing is predicated upon one fact, often not taken into consideration, namely, that allowance has been made—and it must be made whenever a refinement in interpretation is important—for such physiologic causes of leukocytosis as the digestive factor, cold baths, exercise, menstruation, etc. Particularly, if a count of 10,000 is to carry weight in a diagnosis, must the blood be taken with the patient fasting.

Incorrect diagnoses based upon the results of laboratory tests improperly executed are common. We frequently encounter

the diagnosis of tuberculosis made in some obscure condition on the strength of the reactions produced by large initial doses of O. T.; and conversely, tuberculosis is eliminated as a diagnosis after a single small hypodermic injection of tuberculin, instead of after a series of gradually increasing doses.

The newer work in the renal functional domain, in acidosis, basal metabolism, etc., all requires a thorough grounding in chemical methods and a great deal of experience to yield data which permit of diagnostic conclusions. The Wassermann test is carried out by not a few whose lack of training makes the value of their results worthless. One point is simply that the laboratory dabbler does himself and his patients more harm by attempting work beyond his skill than by adhering to the routine methods of physical and laboratory diagnosis reinforced by his clinical experience and good judgment.

What has just been noted as to the danger of amateurish laboratory work applies particularly, in the harm it may do, to tests the value of which is still unsettled. Even the well-equipped technician needs clinical experience and balance to give these their proper weight. We are still far from understanding the value, relative and absolute, of the several renal functional tests; of the Abderhalden reaction; of the fractional method of gastric analysis; of indicanuria, etc., yet how frequently we meet with men who, without calling upon their clinical judgment for the necessary qualification of their laboratory data, speak with assurance of the type of the nephritis and the exact localization of the underlying pathology; of the organ or portion of organ shown to be involved by the Abderhalden method; of gall-bladder rather than gastric pathology because of the curve of gastric acidity; and of every conceivable condition because there is an "increase" of indican in the urine!

Similar considerations are applicable to many of the new signs and symptoms which appear in the literature from time to time, have a passing vogue, and then are heard of no more. Illustrations of the same will occur readily to every one, and may be omitted.

We do not wish to be understood as decrying modern methods

of diagnostic refinement, whether in the field of physical diagnosis or in the laboratory, but only as condemning a too great leaning upon these methods at the expense of the tried routine backed up by clinical experience. A nice perspective is the thing to be desired. Apropos of this we may mention the fact that the most able clinician with whom we have worked—one whose diagnoses even in their detail were verified at autopsy in the very great majority of cases—was one who relied almost exclusively upon the routine methods of examination and who regarded the special methods as lights or shadows in the picture as a whole.

6. We would call your attention, finally, to **the riding of a hobby** as a cause of frequent diagnostic error. It is a widespread fault, common both among general practitioners and specialists. Its dangers are obvious and diverse. The man who is not in the habit of making thorough routine examinations is likely to see in a large proportion of his cases examples of the condition upon which enthusiasm is centered at the particular time; the more thorough man, not finding a cause for the symptoms complained of, turns almost instinctively to his hobby of the moment.

It appears to us that the fault we are discussing could be largely eliminated if medical men of authority in their addresses and writings used a little more discretion in giving to the profession opinions based upon insecure evidence; or, if they wish to gain priority in the particular matter, they should so publish the results of their work as to let the physician at large know that it is still *sub judice*. The general practitioner looks for guidance to the special worker and is likely to follow this guidance with a supreme trust.

We shall forbear giving illustrations of this sixth point. A moment's thought will suggest such to every one of you—views as to medical subjects which held the stage for the moment and then faded out. Their value lies in the fact that they reopen a medical field—perhaps dormant for years—to intensive review; this, at least, clears the atmosphere in that particular field. Their power for harm is considerable, namely, to the

degree in which they delay the establishing of a correct diagnosis and the institution of proper treatment.

These six points constitute, in our opinion, the principal sources of error in diagnosis. In part, they are a matter of lack of training; in part, a matter of lack of thoroughness and of slovenly routine; and in part, finally, a lack of that calm, judicial attitude which cannot be swayed or bent by the enthusiasm of the moment.

CLINIC OF DR. ISAAC A. ABT

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INFANTILE ECZEMA

Etiology; Protein Sensitization; Heredity; Skin Pigmentation; Neuropathic Phase; Vasomotor Disturbances; Metabolic Disturbances; Various Factors in Causation; Clinical Forms; Accompanying Symptoms; Relation of Eczema to Asthma; Complications; Full Discussion of Treatment and Management.

March 13, 1920.

DURING this hour I desire to devote some time to the consideration of infantile eczema. This disease is one of the most difficult and obstinate that we are called upon to treat. The old Continental physicians used to call it "the forties" because it was likely to last forty weeks and sometimes much longer.

The first child to whom I wish to direct your attention is a boy aged eight months. He came into the hospital several days ago with a papular, crusted eruption of the face, scalp, chest, arms, and legs. The condition was first noticed about three and a half months ago. It began on the face and next appeared on the legs, itched a great deal, and consequently the baby was almost continuously scratching. At the onset the child was on the breast and, as so frequently happens, he was weaned because of the eczema, which, I may tell you parenthetically, is a great mistake. These babies may do poorly at the breast, so far as the eczema is concerned, but they nearly always do much worse with various cow's milk mixtures. The baby has had various milk mixtures, and just before entering the hospital was given skimmed milk. The mother tells us that an

older child in the family had a similar eruption when it was eight months old.

The little patient under consideration showed some temperature exacerbations, the highest temperature being 101.4° F. The blood examination showed 6 per cent. eosinophils, though the total leukocyte count was 10,000.

A diet of skimmed milk was ordered, the crusts were removed with olive oil, and Lassar's paste without salicylic acid was very thickly applied and covered with gauze. It is now six days since the patient has been under treatment, and he is so far improved that he is ready to go home today. He has gained 4 ounces during his stay in the hospital.

I have another little male patient, nine months old, whom I wish to show you. He entered the hospital with a temperature of 104° F. and a severe generalized eruption. The skin of his face was rough and harsh, with marked excoriations and crusts. A similar condition was present on the extremities, though not so severe. In many of the affected parts a secondary infection had occurred. This was particularly true in the hairy scalp in the occipital region. Moist boric dressings were applied to the head and pustular areas. Subsequently moist aluminum acetate dressings were employed. After the skin had become softened and the acute inflammatory condition had subsided, the usual treatment, consisting of the application of Lassar's paste without salicylic acid, was instituted.

The child has been in the hospital for six days. It has been necessary at times to give sedatives because of the extreme restlessness, itching, and consequent scratching. The temperature remained over 102° F. for the first few days, but has now subsided. The blood examination showed a leukocyte count of 28,000; there were 4 and 5 per cent. eosinophils on two different occasions. The diet was restricted to skimmed milk, cereals, and vegetable broth. At present there is a normal temperature and a smooth, soft skin, which would hardly indicate the severe involvement which he presented upon admission.

Let me show you another child very briefly. This patient is a little girl one year old who was brought to the hospital several

days ago with an erythematous, papular, slightly crusted eruption about the face and eyes. The itching was intense. Neither mother nor child had slept for weeks, and the mother, in desperation, brought the child to the hospital.

The eruption began two months ago as an erythematous rash. At home the feeding consisted of various milk mixtures with cereals. Skimmed milk was substituted for the ordinary milk. Cold cream and Lassar's paste without salicylic acid were applied locally, and 1-grain doses of veronal were given at night because of the extreme restlessness.

You notice the child this morning. The rash is disappearing; she is playful and happy; the itching is less severe; she is taking food; and in a day or so will be ready to go home.

All of our cases are not so simple—all do not yield so readily to treatment. But let us consider in a general way this entire subject. The older doctors considered eczema due to some constitutional anomaly. Indeed, this was in accord with the older pathology that disease was largely due to disorders in body fluids. These disorders were called "dyscrasies," and this was the pathologic trend in the days when the so-called "humoral pathology" held sway. These views were changed with the advent of Virchow, who no longer saw any basis for humoral pathology, but viewed disease as an alteration in the tissues and cells. He studied an organ and found in it certain degenerative or inflammatory processes and, consequently, classified disease on the basis of cellular changes in certain organs. The new school saw nothing of importance in individual constitution. Even the new science of bacteriology, which soon followed, made no allowance for constitutional anomalies. In recent times renewed attention has been given to the individual himself, his structure, his constitution, his individual resistance against disease-producing influences. Not all individuals are alike regarding their resistance against disease, their susceptibility to illness, or their reaction to disease when they are attacked. The same disease may lead to a very severe and fatal illness in one individual and run a very mild course in another. Suffice it to say that the older physicians of the "humoral pathology" period

laid too much stress on the importance of the constitutional factors, while the modern clinicians, who are under the spell of cellular pathology and bacteriology, underestimate the constitutional element.

We think that certain cases of eczema occur because the infant has inherited a certain type of constitution. We speak of this condition as a "diathesis," and we understand by diathesis a congenital tendency to disease not starting in any particular organ, but manifesting itself in various parts of the body and producing a variety of clinical symptoms. Now when this diathesis is present in a certain individual, in this case in the body of a baby, a moderate irritation which would not produce disease in the ordinary infant produces a skin lesion in an infant who has the given diathesis. While this matter of diathesis has been relegated to the background by some of the great medical leaders, nevertheless the English, French, and Italian physicians have invoked this theory to explain a variety of disorders. In 1905 Czerny reintroduced the term into pediatric literature with particular reference to the exudative diathesis and eczema.

Etiology.—The various etiologic factors in the production of eczema are not clear. In general, we may consider two causes: first, the individual or internal, largely constitutional, congenital anomalies; second, irritations and injuries. One may speak of constitution and predisposition, on the one hand, and determining or provoking causes on the other. Certain authors would include exudative, lymphatic, and arthritic diatheses, the arthritismus of the French, under predisposing causes. Gout, diabetes, and obesity predispose to eczema. The last is associated with perspiration, which acts as a skin irritant.

Protein Sensitization.—Recent investigations have shown that eczema tends to occur as a result of a specific kind of food. Because of the close relationship between eczema and other manifestations of allergy the phenomenon has been recently studied from the standpoint of food sensitization. Blackfan found that only one infant of a series of normal children under one year gave a positive reaction to egg white. This child gave negative reactions to cow's and mother's milk. It was a nine-

weeks-old infant who had been on the breast entirely. Of 27 eczema babies, 22 gave positive reactions. The importance of these observations is somewhat dimmed by the subsequent findings of Greer, who confined his studies to infants with gastro-enteritis and atrophy. Of 26 cases, 23 gave positive reactions with lactalbumin and 3 with casein. Talbot reports the routine skin tests on 100 unselected cases in which only one positive result was obtained. He finds that not all eczema cases give protein sensitization results. One of his cases gave no reaction, though 60 different proteins were used. On the whole, the studies on anaphylaxis have been disappointing, and little additional data have been gleaned concerning the etiology or treatment. It would seem that the problem should be approached both from the standpoint of the baby who has had nothing but breast milk, and who, therefore, has no ferments elaborated for other foods save what might have been transmitted by mother's milk, and from the standpoint of the artificially fed baby who has had other foods in addition to cow's milk.

Drugs.—Some babies show an idiosyncrasy toward certain drugs. For example, antipyrin produces a marked skin reaction in some instances.

Age.—Moro and Kolb found the disease began most frequently in the third month. Cases occur with frequency during the first year and begin to diminish after the second year.

Sex.—Among young children the disease seems to be more common among little boys. Moro and Kolb found 75 per cent. among little boys. In passing it might be mentioned that many diseases during early life seem to attack boys more frequently than girls. The reason for their high mortality is, therefore, apparent.

Heredity.—Most frequently the history shows that one of the parents has suffered from eczema or some other symptom of exudative diathesis. Very frequently brothers or sisters are affected with the disease. Some authors maintain that baldheadedness of the father indicates a tendency to eczema which shows itself in the offspring. The French say that seborrhea of the scalp is relatively frequent among arthritic adults,

and consequently the children from such fathers are hereditarily predisposed to eczema. They also maintain that the arthritic diathesis and eczema occur more frequently in the children of the well-to-do and those who live well. One comes to the conclusion that the factor of heredity cannot be founded upon statistical information.

Skin Pigmentation.—Blondes have more delicate, irritable skin than brunettes, and for this reason it is considered that blondes are more frequently affected. Recently Moro and Kolb showed that the majority of infants affected with eczema had white skin, blonde hair, and blue eyes.

Neuropathic Phase.—According to Czerny's view, neuropathic conditions are often combined with exudative diathesis. Itching will be mild or severe, depending on the condition of the nervous system. He points out that among well-to-do families, where spoiled children abound and the parents are of the anxious type, marked nervous symptoms occur among eczematous children. Numerous authors have noticed that bronchial asthma more often occurs with eczema of the better classes of people.

Vasomotor Disturbances.—Eczematous children show an increased vasomotor excitability. One investigator attempted to show this by breaking the skin with a Pirquet borer without using the tuberculin. In such children marked reaction occurred to this slight trauma. He also found that eczematous children who were vaccinated for small-pox showed a more pronounced reaction than those not so affected.

Metabolic Disturbances.—There is some difference of opinion among various writers as to the influence of nutritional disturbances in producing eczema. Metabolic disturbances associated with disturbances of internal secretions may produce skin changes. In some women eczema is one of the first signs of pregnancy. Many women have recurrent eczema at the menstrual epoch, and a certain number show seborrhea of the scalp and cheeks. It has been maintained that the condition disappears when thyroid extract is administered. Ravitch and Steinberg point out that mothers who suffer from dysthyroidism during preg-

nancy and lactation bear children who frequently have eczema. These authors assign an important influence to the fat metabolism, and they find that infantile eczema is induced by an alteration of thyroid secretion.

Alimentary Tract.—Dyspepsia and alimentary intoxication may occasionally produce eczema, though sufficient proof is not at hand to establish their close causal relationship. The same is true of constipation.

Bacteria.—It is the consensus of opinion that the occurrence of bacteria is only secondary. Staphylococci are commonly found in all of the eczemas.

Dentition.—The old story of the relation of dentition to eczema need not be discussed. I have never been convinced that dentition had any influence on the occurrence of eczema.

Infectious Diseases.—One frequently notices that eczema temporarily disappears during an acute infection. This holds true for pneumonia and the severe intestinal disturbancess associated with diarrhea. Measles and chicken-pox frequently aggravate a case of eczema. A vaccination pustule sometimes becomes increased in intensity in eczematous children.

Dietary.—Overfeeding with milk, whether mother's or cow's, plays an important rôle in the production of this condition. It is not certain what milk factor produces the eczema. Finkelstein thought that excessive salts were responsible, while Feer thought that the condition resulted from the combination of too much salts with fat. Many believe that the fats alone are responsible. Significance of milk as an etiologic factor is well shown by the fact that when a mixed diet is given eczema tends to disappear. Czerny has emphasized the fact that next to milk, eggs tend to produce the condition. Feer maintains also that meat broth leads to the production of eczema. Many clinicians limit the milk and egg intake and increase the starchy foods. Many children who are overfed suffer from this disorder, but it cannot be denied that many children with eczema have not been overfed and have received proper quantity and quality of food.

External Factors.—Little need be said concerning the external exciting causes. We read in every text-book that they are mechanical, chemical, thermal, and electrical. It need only be mentioned that too frequent bathing or the use of irritating soaps may produce eczema, particularly in children with delicate skins. Uncleanliness, irritating secretions, such as perspiration, vomitus, diarrheal stools, and acid urine, tend to produce intertrigo, though even in these cases predisposition seems to play an important part. In some babies the skin is unaffected even if it be allowed to lie in strong urine or fecal evacuation night after night. I have sometimes seen a severe intertrigo in children when the diapers had been washed in caustic soap, ammonia-water, or washing powder, and had not been sufficiently rinsed.

Clinical Forms.—Moist, crusted eczema occurs in fat, healthy looking children. It begins on the scalp or face, may be confined to the head, or spread to the neck, trunk, arms, and legs. This form occurs particularly in children overfed with milk. Constipation is frequent in these children. Spontaneous recovery usually occurs at the end of the first year when a mixed diet is instituted.

Dry, disseminating eczema occurs mostly in artificially fed, pale babies suffering from nutritional disturbances and diminished weight. Constipation may be present, although diarrhea is more often the case. This type consists of distinct, dry, red, desquamating, infiltrated islands and also of papular and pustular foci. Weeping is moderate.

Scrofulous eczema occurs on the face and mucous membranes of the eyes, ears, and nose. Phlyctenular keratitis, rhinitis, and swelling of the upper lip and glands of the neck are likely to occur in this variety. In many of the cases a positive tuberculin test is found. So far as the occurrence of phlyctenules is concerned, they are found with a tuberculous process, but may be present without tuberculosis.

Intertrigo is a frequent form of infantile eczema and is often the result of irritating secretions and excretions, such as the urine, stool, and perspiration. It is frequently accompanied by great

itching and often occurs in excessively fat children. The eruption is located in the folds of the skin in the inguinal, axillary, and pubic regions.

Erythrodermia desquamativa is discussed by Liener of Vienna. The principal symptoms of this form are marked seborrhea of the scalp and redness of the skin of the entire body, together with desquamation occurring in large scales. This disease attacks delicate undernourished children in the first three months of life suffering from gastro-intestinal disturbances. Many of these children have been breast fed, and Liener thinks that the disease occurs on a constitutional basis. In one instance the disease occurred in 4 brothers and sisters, and in another in twins. In one case the rash suddenly disappeared and was followed by a severe diarrhea leading rapidly to death. Skimmed milk and skimmed breast milk seem to relieve the condition markedly.

Strophulus, also spoken of as lichen urticatus, has only a slight relation to eczema. The lesions consist of hard, markedly itching, red nodules which locate themselves along the trunk and thighs. Czerny thought that strophulus was closely related to the exudative diathesis. Feer thought that the children suffering from strophulus possess a constitutional anomaly and that certain dietetic violations produce the lesions. Excessive amounts of milk and eggs during the first three years of life and constipation play an important rôle. In these cases a mixed diet of vegetables and fruit tends to cause improvement. Frequently it is difficult to ascribe any cause.

Accompanying Symptoms.—We need not speak of the common symptoms, as pruritus, increased vasomotor excitability of the skin, or the symptoms on the part of the mucous membranes. One frequently sees the geographic tongue, which Czerny considers a most frequent and important symptom of exudative diathesis. Many of the children also have catarrhal and inflammatory conditions of the nose and larger bronchi. Tonsillar hypertrophy is frequent.

Relation of Eczema to Asthma.—In the clinic one is struck by the frequency of asthma in children who had eczema in in-

fancy. It is noticeable that the asthmatic condition manifests itself after the eczema has disappeared or healed. Eosinophils are frequently found in the blood and stools. The eosinophilia in eczema reaches as high as 33 per cent., and as the eczema disappears the eosinophils diminish.

Complications.—Gunn and Porter describe 3 cases of nephritis following eczema. Several cases have been reported after the impetiginous form of eczema. The disease in these cases is infectious, and metastases occur by reason of the streptococci and staphylococci present in the blood. In considering nephritis we must remember that some of these cases are really due to chemicals applied to the skin—beta-napthol, tar, phenol—which are absorbed from the surface.

Eczematous Death.—Not a few suddenly fatal cases have been reported in children who have been apparently in fair health though suffering from eczema. These cases of sudden death may be explained on the basis of status lymphaticus, though the pathologic position of status lymphaticus is not secure. Some writers think that obesity bears some relation to sudden death. On the other hand, there are some very excellent pathologic opinions on record which would tend to assign status lymphaticus and enlarged thymus a definite position in the causes of death in eczematous babies as well as in older individuals. Feer adheres to status lymphaticus as the cause of sudden death, and says that in acute infectious diseases, especially diphtheria and scarlet fever, death may occur with insignificant objective findings. He cites a case in which the child died suddenly after it had taken its meal with relish, without showing any symptom pointing to organic disease. We have here a case of an eczematous child dying of circulatory failure. Autopsy showed hyperemia of the lung or beginning bronchopneumonia. In other cases these eczematous infants present a high temperature, dyspnea, cyanosis and somnolence, followed by death within twenty-four hours. Some authors have attempted to show that sudden death in eczema is due to foudroyant sepsis. In some cases the findings are those of an acute infection or a severe intoxication and give the impression sometimes of anaphylactic

shock. Indeed, the question has been raised whether this late form of eczematous death is not one of anaphylaxis.

Treatment.—The treatment may be divided into general, dietetic, and local. As far as the general treatment is concerned, the most important consideration is the feeding, not only as regards quantity, but the kind of food. For a long time clinicians have restricted the quantity of milk. Bohn suggests that bouillon and cereals be substituted for the milk. However, Feer saw an injurious effect from animal broth. Many eczematous children take too much food. Czerny pointed out the ill effects of eggs and milk on eczema in 1905, and suggested the substitution of fruit, vegetables, and cereals. For older children he permitted a small quantity of meat. He considered the fat of milk particularly injurious. Along these lines Feer found skimmed milk or even whey was useful in the diet of infantile eczema. Finkelstein thought that eczema was produced by excessive salt content in cow's milk. For this reason he freed the milk of its salt by removing the curds from the whey and substituting barley-water for the whey content of the milk. A good many observers found that babies fed on this milk lost in weight, and it was impossible to properly nourish the child by this food. When the quantity of milk is diminished, the deficiency may be supplied by cereals even in the second and third month. From the sixth month various forms of cereals and vegetable soups may be employed; fresh fruit juices from the third to the fourth month; vegetables and stewed fruit from the sixth month. When eczema occurs in the breast-fed child one or two breast feedings may be substituted by cereals. After the second year the milk may be materially reduced and the vegetable diet largely substituted. Constipation should be overcome, and this may often be accomplished by generous quantities of fruits and fruit juices.

A recent paper of Langstein's is of more than passing interest. He points out that if certain foods were the sole cause of eczema their withdrawal should act in a specific way by curing the skin lesion. He finds, however, that this is not true. It seems natural to omit any foods which give a positive skin test. Since milk heated to 240° F. no longer causes anaphylaxis in sensitized

animals, it has been suggested to give boiled milk. Talbot also suggests desensitization by treating the milk with lactic-acid-forming bacteria or giving rectal injections of milk.

Children suffering from eczema should be protected from infections and from overstimulation mentally. It is uniformly noted that children who are excessively nervous have a marked pruritus and that they scratch more than those of more stable nervous constitution. Thyroid extract has been recommended, particularly in children with dry skin and seborrheic eczema, such as occurs in fat children. Cod-liver oil and iodid of iron have been recommended, and arsenic is still used in some quarters. The alkalies are recommended by various clinicians. Stoeltzner recommends citrate of soda. Cathartics have been employed in the treatment principally on the supposition that they diminish intestinal intoxication.

Roentgen rays have been used in both the dry and the moist as well as in the infectious and seborrheic varieties. The rays are applied two to four times at intervals of ten days. A pause of four weeks is allowed, followed by one or two more treatments at long intervals. Jadassohn feels that he has succeeded in curing a considerable number of cases by this method.

Amyl nitrite inhalations have resulted in quick recovery from exudative eczemas, according to the report of Berend. In determining the dosage he found that 1 drop gives no effect, 2 drops reduces the redness of the skin considerably when inhaled in thirty seconds, and 3 drops inhaled in thirty seconds gives a good effect. He says that larger doses, 5 to 10 drops, may be used if the time is reduced to a momentary inhalation. He states that young children with normal distribution of blood in the surface vessels are very much benefited by this treatment. Cases with exudative diathesis react by reddening of the skin followed by almost immediate blanching, particularly noticeable in eczema.

While the careful regulation of the diet, recognition of the factors of general treatment, and observance of the rules laid down by Czerny and his school are of value in the treatment of eczema, nevertheless, a well-planned local treatment is nearly

always necessary to cause a subsidence of the eczema and its attending pruritus. Little need be said about the local treatment, as this form of treatment belongs more to the dermatologist than to the pediatrician. In our experience several points are of importance in the management of these cases. A bland, non-irritating ointment should be employed. Lassar's paste without salicylic acid, zinc ointment, or ordinary cold cream may be used in ordinary cases. The salve should be applied thickly to the afflicted part, and it should be so applied that it will be retained in place. On the extremities the applications may be made on lint or cloth and bandaged into position. For the face we have found a mask very useful, which leaves openings for the nose, mouth, and eyes. The children are not at all disturbed by such an application. The crusts should be removed by the liberal use of olive oil or cold cream, and for this purpose the ointment may be applied on lint, gauze, or gutta-percha tissue, and retained in place six or eight hours. The skin should then be washed with olive oil and dried with almond meal. In the infected cases one may use moist boric dressing until the acute reaction is passed. Soothing lotions of lead may then be used, to be followed by a bland ointment.

SYMPOSIUM CLINIC. DEPARTMENT OF DR. ISAAC A. ABT

MICHAEL REESE HOSPITAL (SARAH MORRIS MEMORIAL HOSPITAL FOR CHILDREN)

DR. TUMPEER: THE SCHICK TEST.
DR. DYSON: PYELITIS IN CHILDREN.
DR. GERMANN: HIGH SUGAR FEEDING.

March 6, 1920.

DR. ABT: I know you will all be delighted this morning when I tell you that I, personally, shall bore you but for a few moments. Some of the younger men have been studying groups of cases in the wards, and at my invitation they will present their findings and results to you. You will find that the reports are purely clinical, and the more scientific laboratory reports and work will be reserved for another time.

The first report will be on the Schick test as it has been conducted in our wards, and Dr. Isidor Tumpeer will tell you about the technic of the method, its uses and results. He will also tell you about the toxin-antitoxin mixture and its uses.

THE SCHICK TEST

DR. TUMPEER: In connection with diphtheria immunity I wish to discuss the Schick test and the toxin-antitoxin mixture. The Schick test is applied to determine the reaction of the individual to diphtheria toxin and informs us whether the individual tested is immune or susceptible to diphtheria. A positive result means that the subject is susceptible, and a negative test that he is immune to diphtheria.

To carry out the test diphtheria toxin is so diluted that 0.2 c.c. contains 0.02 of the M. L. D. The control consists of some of the

identical sample of toxin heated for five minutes at 75° C. The purpose of the control is to exclude the reaction that sometimes occurs due to the trauma of injection and the protein of the bacterial bodies. It must be remembered that the reaction sought is that of the toxin itself. The broth filtrate of the diphtheria culture contains not only the exotoxin produced by the bacilli, but some of the autolyzed bacterial bodies which in certain individuals are capable of producing a protein reaction of their own. By heating a portion of the toxin preparation to be used to 75° C. for five minutes the toxic fraction is destroyed, leaving the proteins of the bacterial bodies and culture intact. Since one preparation of toxin varies from another in the amount of extratoxic protein it is absolutely essential to prepare the control mixture from the identical preparation of toxin for any accurate determination.

The test is an intracutaneous one and is made commonly on the volar surface of the forearm. One arm is used for the toxin and the other for the control, although both may be placed on the same arm, leaving enough distance for the development of the reaction. The needle should be long and fine, and a short bevel at the end is preferable, so that you do not puncture the skin when you bring the end of the needle to the surface. We find the ordinary Von Pirquet syringe satisfactory, since 0.2 c.c. can be accurately measured. The needle is inserted into the skin and the end brought beneath the epidermis so that the mouth of the bevel is plainly seen under the skin. Some men keep the needle in the corium all the time they are inserting, while others puncture the subcutaneous tissue and then come out to the surface. If the needle has been properly inserted a wheal will be formed by the injection in which the mouths of the sweat glands will form little pits. Koplik and Unger have devised another method of performing the test in which undiluted toxin is used. A hypodermic needle bent at an angle of 170 degrees $\frac{1}{4}$ inch from its end is dipped into the undiluted toxin and inserted intradermally. The toxin is left behind as the needle is withdrawn. This method has the advantages of simplicity and greater practicability. The authors state the traumatic pseudoreactions are obviated and that 75 per cent. of the anaphylactic pseudo-

reactions are eliminated. Otherwise the results check accurately with the test as carried out by the Schick technic. Inasmuch as the pure toxin retains its potency for one year when kept on ice, it is more available than the diluted material, which must be made up in fresh solution and has to be accurately measured. The Koplik and Unger method is practically painless.

The reaction obtained by the Schick test may be (1) true, which may be (*a*) positive or (*b*) negative, (2) pseudo, or (3) combined. If no reaction develops at the site of either inoculation in forty-eight hours the test will most probably be negative. At the end of ninety-six hours no reaction spells a negative test. If there is a reaction around the toxin and none about the control in forty-eight hours the test will be positive. This becomes very pronounced at the end of ninety-six hours. The infiltrated area reaches the size of a quarter. It changes color, becoming brownish, and ultimately desquamates. The brown tinge persists several weeks. The pseudoreaction consists of an infiltration about both the toxin and control and appears within a few days. They fade together and have usually disappeared by the end of ninety-six hours. The combined reaction occurs in an individual who is susceptible to both the control and the toxin. There appears a similar change about both sites of inoculation at the end of forty-eight hours, but the control reaction will have faded by the end of ninety-six hours, while the reaction about the toxin will still be in evidence and will follow the usual course of such a reaction, with scaling and discoloration.

The Schick test finds its greatest use in determining the susceptibles in the presence of an epidemic. Of course, after they have been found, the practice differs in different places. Some give an immunizing dose of antitoxin to all those showing a positive test. Others believe in segregating the susceptibles and watching them closely for the development of clinical diphtheria, at which time a sufficiently large curative dose can be administered. I believe that the latter method is the preferable one where you have strict control over your patients and where you can carefully observe them. Among other things it saves the promiscuous sensitization of individuals to horse-serum.

It seems to me that the principal use of the Schick test from now on will be its employment with the view of determining the susceptibles, so that the active immunization may be carried out with the toxin-antitoxin mixture. Contagious disease hospitals find it convenient to test their incoming physicians, nurses, and attendants who will come in contact with diphtheria patients. Quick immunization of those giving a positive test may then be carried out. The test is also of some value from a suggestive standpoint in the diagnosis of obscure paralyses and conditions which may be postdiphtheric. If such a case shows a positive test the chances are that the exciting condition was not diphtheria, because convalescent diphtheria patients usually give a negative test.

The reliability of the test is sometimes called into question when an individual who recently gave a negative Schick develops diphtheria. This circumstance is exceptional, but when it does occur one should look to the materials with which he carried out the test. One must not lose sight of the fact that immunity is a changing factor and that an individual may lose his immunity under conditions causing a general lowering of resistance. The other possibility should also be considered. An individual, particularly one whose resistance has been lowered, may receive an overwhelming infection, which breaks down or overcomes his immunity for the ordinary infection. Unger, of Chicago, has recently reported a series of 25 cases of typhoid and paratyphoid fever which occurred in troops inoculated against these diseases. You can think of the situation in this way. The gas masks were proof against the gas cloud attacks and the usual concentration from gas shell fire, but even the mask failed to protect against the intense concentration of gas from a shell which had exploded in the immediate vicinity. In children up to two years susceptibility mounts from 15 per cent. at three months to 70 per cent. at two years. You can see how quickly the Schick response will change if taken at different times during that period.

THE TOXIN-ANTITOXIN MIXTURE

The toxin-antitoxin mixture is so prepared that the toxic properties are destroyed without impairing the power of the toxin to stimulate the tissue cells of the body to produce antitoxin. This method of immunization is not a vaccination in the strict sense of the term. Instead of the bodies of the bacteria forming the antigen, as is the case with those organisms which produce their results by an endotoxin, we have to deal with an exotoxin which must be used as antigen. The administration of the toxin-antitoxin mixture is a method of active immunization against diphtheria. It differs from the passive immunization in its slower development, but it is more persistent.

It is interesting to note that this development in the immunology of diphtheria arose from the observation that guinea-pigs who survived test amounts of antitoxin after intoxication required more toxin to kill them subsequently.

In order to understand the preparation of this mixture it may be well to recall a few definitions which occur in this connection. You will remember that the M. L. D. is the smallest amount of diphtheria toxin necessary to kill a 250-gram guinea-pig. The antitoxic unit is that amount of antitoxin which will just neutralize an M. L. D. The L plus dose is that amount of toxin necessary to kill such a guinea-pig when 1 unit of antitoxin has been administered. In the preparation of the most effective mixture Zingher points out that at first an overneutralized mixture was used. This contained 1.5 to 2 units of antitoxin per L plus dose. Later a neutralized mixture was used. This contained 1.25 to 1.5 units of antitoxin per L plus dose. As time went on, however, they found that the most efficient mixture was a slightly underneutralized mixture containing 1.125 to 1.25 units of antitoxin per L plus dose. After such a mixture is prepared it is tested for potency by injecting 1 and 5 c.c. respectively in two guinea-pigs. Slight induration occurs about the 1 c.c. injection. About the 5 c.c. quantity there should be moderate or marked local induration and late paralysis. The animal should not die acutely of diphtheria poison. If he

dies within five days it indicates the presence of free toxin, which must be carefully neutralized.

The administration of the mixture is carried out in three injections at weekly intervals, according to Park. Each injection consists of 1 c.c. administered subcutaneously at the insertion of the deltoid. In infants under one year 0.5 c.c. is given. There is no general reaction even in infants four days old, according to the members of the New York Department of Health, who have done the most work with this method.

The result of this active immunization is shown by the following figures. In a few weeks a small percentage have developed negative Schick tests:

Age.	Per cent.
2 months	80
3 months	96
4 months	98

The other 2 per cent. may be rendered immune by one or two subsequent injections. The method of giving but one injection gives about 60 per cent. of negative tests; two injections give 75 per cent.; and three, as we have seen, give 98 per cent.

To understand the application of toxin-antitoxin let us consult the tables of Park on susceptibility by the Schick test:

Age.	Per cent.
Under 3 months	15
3 to 6 months	30
6 months to 1 year	60
1 to 2 years	70
2 to 3 years	60
3 to 5 years	40
5 to 10 years	30
10 to 20 years	20
Over 20 years	15

Infants show immunity because of the transmission of immunity from their mothers, 85 per cent. of whom are immune according to Park's table. In other words, infants under three months show the same immunity as adults over twenty years. The greatest diphtheria mortality occurs at two years, and this

corresponds closely with the greatest susceptibility as shown by the Schick test. Because of the rapid development of susceptibility of infants and young children Zingher advocates immunizing all infants under eighteen months without determining their response to the Schick test. After eighteen months he performs the Schick test before giving the mixture. It is very readily seen that if you tide the child over the third year the worst period is avoided, and from that time on his immunity will rapidly increase, as the tables illustrate. The results with toxin-antitoxin administration have shown that at the end of three years the actively acquired immunity has still persisted. How long such immunity will last is being watched with great interest.

The application of the active immunization so far has been largely in some of the children's homes in New York City, and only here and there is it used in the general population. It will require education of the public before the test is as widely adopted as, for instance, small-pox vaccination. For quick results in the presence of an epidemic we should again emphasize that the toxin-antitoxin mixture is not effective because it takes a few months before the results are seen. In such cases the susceptibles should be detected by the Schick test and given an immunizing dose of antitoxin, or where the conditions are ideal the exposed individuals should be segregated and watched for signs of clinical diphtheria, at which time a curative dose should be administered.

Dr. Abt: Dr. James E. Dyson, one of the workers in our clinic, has given particular attention to the comparative study of the treatment of pyelitis, and he will report to you about a series of remedies which he has used and results which he has obtained in the treatment of acute and chronic pyelitis in infants and older children. We have not had a large number of cases, but small groups have been studied intensively to test out the effect of certain drugs and vaccines.

PYELITIS IN CHILDREN

Dr. Dyson: I wish to present the results of a study of pyelitis in children with particular reference to a comparison of different methods of treatment. The early diagnosis and subsequent management of a case of pyelitis or pyelocystitis depends on a relatively exact method of counting the cells and estimating the bacteria and pus clumps in the urine. The methods in common use of counting the cells in a low-power field, or of centrifuging the specimen, decanting off the fluid, and counting the cells in the sediment under a high-power field are all subject to great variations. A more exact method by using a blood-counting chamber may be standardized. We use a drop of the specimen that has been thoroughly disturbed to assure an equal concentration of the sediment. Fill the blood-counting chamber, allow the blood to settle, and count the cells in a square millimeter. It is more accurate to count 4 squares and take an average. In this square millimeter of $\frac{1}{10}$-mm. thickness we are using a constant amount of the specimen and get results which can be compared to the standard normal.

In this study we have found the average count of white blood-cells in normal boys to be 2 to 3 cells per square millimeter and in normal girls 3 to 5 cells. In a group of heart cases the count in the boys ran from 3 to 5 and in girls from 5 to 9 cells per square millimeter. In a group of nephritis cases the boys' count was 5 to 12 and the girls' 5 to 35. Another group of cases with an increase of cells are the acute infectious diseases and intoxications. In a large number of cases of influenza pneumonia 4 to 12 cells were found. One pneumonia (male) case with constant temperature of 103° to 104° F. only had 2 to 4 cells for days. One influenza case (female) had 214 cells, probably a latent pyelitis which became active during the intoxication of the influenza.

The object of this study is not only to determine the normal cell count in the urine of healthy children, but to find the number of cells present in disease. The gravity of pyelitis can be estimated by knowing the quantity of pus and bacteria present in

the urine. Treatment can be more intelligently managed by a knowledge of the daily variations of these pathologic elements.

Case I.—According to the British method of treatment by giving alkalies, we gave the first case, an eight-year-old girl, potassium citrate, gr. x, four times a day. The urine had an acidity of 14 to 22 c.c. N/10 Na OH. In three days we increased the potassium citrate to gr. xv four times a day and the urine was still normal, acidity of 18 c.c. N/10 NaOH. Two days later gr. xx, three doses, were given in the forenoon, and the urine that afternoon was alkaline to litmus, but still acid to phenolphthalein, neutralizing N/10 NaOH. The urine during this period was packed with pus-cells, many in clumps. Their number varied somewhat from day to day, but showed no general diminution. There were many bacteria in all specimens and no clumping in the alkaline urine.

To test the statement made by some observers, that changing the reaction of the urine from alkaline to acid causes agglutination of bacteria and improvement of the pyelitis, we gave sodium acid phosphate, gr. v, four times a day, increasing to gr. vij every hour for ten doses, for two days, and later gr. xv three times a day for ten days. During the administration of sodium acid phosphate the urine did not show a consistent increase in acidity; a few examinations did show slightly higher acid contents than normal. No difference was seen in the number of pus-cells or bacteria. Special notice was made of the arrangement of the bacteria for agglutination, to determine by what treatment bacterial clumping could be effected and to note the effect of such clumping upon the course of the disease. There was slight clumping of bacteria in three specimens with an acidity of 20 and 22, and marked clumping in one specimen of 23 acidity. Some 32 examinations ranging from an acidity of 3 to 47 were made, and as many specimens of 20 to 23 did not agglutinate as did. No clumping was seen in the highest acidity or the lowest acidity specimens.

Guaiacol, ♏iij, three times a day was given by mouth, and urine examination showed guaiacol excreted the first five days of the treatment. After a week of the treatment the pus count

was 262 cells, 5 pus clumps, and very many bacteria per square millimeter. One week later the count was 98 pus-cells, 3 clumps, many bacteria, slight agglutination. One week later the pus-cell count was 36, 1 pus clump, and a few bacteria. At this time the guaiacol was increased to ♏iv three times a day, and one week later pus count was 22 cells, 2 pus clumps with many bacteria, one week later 19 pus-cells, 1 pus clump, and very few bacteria. During this period there was a marked improvement in the patient as well as the urine.

Beginning about ten days later we gave 3 quarts of water a day beside the fluids of her meals, and during the next three weeks the urine became normal. Repeated examinations were made, with cell counts of 3 to 8, no clumping, and no bacteria. The patient's strength and color came back with her health. It is difficult to say what part the guaiacol played in clearing this case. Undoubtedly the ingestion of large amounts of fluid helped in the treatment.

Case II.—The next case I wish to present is a pale, poorly nourished little girl of four years, with a marked kyphosis, who has had a chronic pyuria. She was given benzoic acid, gr. v, three times a day for twelve days, with no increase in acidity of urine nor improvement in cell or bacteria count.

Potassium citrate was administered subsequently, gr. x, four times a day for eight days. The acidity of the urine was reduced as low as 3.5 c.c. N/10 NaOH. The number of pus-cells was somewhat lessened. The bacteria were not reduced in numbers and were agglutinated in but one specimen. This had an acidity of 10 c.c. N/10 NaOH.

Sodium acid phosphate, gr. v, three times a day was then given for two weeks. This treatment increased the acidity of the urine in some specimens, but did not materially affect the number of leukocytes or bacteria. There was no agglutination in high acidity. One specimen of acidity 19 c.c. N/10 NaOH showed some clumping of bacteria.

Guaiacol was started in ♏j three times a day and increased in three days to ♏iv three times a day. This was continued for ten days, when it was discontinued upon the appearance of a

few red blood-cells in the urine. The positive blood test was present but four days and has been absent ever since. During guaiacol administration there was a material lessening of the number of pus-cells and pus clumps in the urine. Bacteria were variable in numbers and showed almost a constant tendency to agglutinate. The acidity during this period ran from 10 to 34 c.c. N/10 NaOH. There was no decrease noted in number of bacteria present during this period of clumping.

Salol, gr. ij, and urotropin, gr. j, five times a day, were given for three weeks. The number of pus-cells varied widely from day to day, but no general improvement was noted. The bacteria were not reduced in numbers, nor was there any tendency to agglutination.

Cystoscopy was done and normal urine was found coming from the left kidney, but the ureteral opening of the right side was inflamed and the catheter could be passed only 1½ cm., which goes to show the impractical side of local treatment for these cases. It is noteworthy that guaiacol in this little patient effected some improvement, while urotropin and salol were ineffective.

Case III.—Our next patient is five weeks old, with pyelitis, loss of weight, diarrhea, and stomatitis. The temperature is 97.4° F. There are six watery green stools a day. Dietetic treatment checked the diarrhea, and potassium citrate, gr. v, three times a day rendered the urine alkaline and reduced the number of pus-cells and bacteria. Although the general tendency was toward improvement, an occasional very high pus-cell count was noted. The potassium citrate was increased to gr. x three times a day for one week. The number of pus-cells was reduced, and they no longer appeared in clumps. No bacteria was found at the later examination. Potassium citrate in this case gave a good result.

Case IV.—The next is a five-month female child with a feeding disturbance, in whom the urine examination showed many pus-cells, many bacteria, and acid reaction. Potassium citrate, gr. v, three times a day and large quantities of fluids were given. The urine was free from bacteria, and yielded a normal cell count

and an alkaline reaction in four days. On the ninth day there was an increase in pus-cells and acid reaction. Potassium citrate was increased to gr. x three times a day. Water was given, 3 ounces every three hours, and the urine became normal and free from bacteria again.

Case V.—This is a two-year-old girl with pyelitis who has been under treatment some time. A catheterized specimen contained great numbers (416) of pus-cells and clumps and many bacteria. Culture contained colon bacilli. During her stay in the hospital without any treatment but light diet and rest in bed her urine improved somewhat. A vaccine was prepared from the colon bacilli, 1,000,000,000 per cubic centimeter. She is returning to the dispensary for vaccine, and after a small injection of 2 minims returned in one week to receive 4 minims, and has been getting two injections a week of increasing dosage up to 8 minims. On examination the urine is not improved in number of pus-cells; however, the last two specimens had fewer bacteria.

Case VI.—The last case I wish to discuss is a female infant of nineteen days, normal feeder, in whom a voided specimen contained a great many pus-cells (228) and many clumps. Examination demonstrated the fact that the patient had a purulent vulvovaginitis, which is not at all uncommon in the newborn. A catheterized specimen showed 21 cells per square millimeter, no clumps, no bacteria. Potassium citrate, gr. v, three times a day rendered the urine alkaline and reduced the cell count to 9 per square millimeter.

Conclusions.—1. Potassium citrate, gr. x, three times a day will lessen the acidity of the urine, rendering it alkaline to litmus. A lessened number of pus-cells and bacteria were observed in alkaline urine.

2. Sodium acid phosphate increases the acidity, but does not improve the urine in pyelitis.

3. Benzoic acid, gr. v, three times a day does not increase the acidity of the urine nor affect the number of pus-cells or bacteria.

4. Bacteria were not agglutinated by high acid or alkaline reactions in the urine.

5. There was rather constant clumping of bacteria during administration of guaiacol, ♏iv, three times a day.

6. There was no apparent decrease in number of bacteria during the period of agglutination.

7. Guaiacol caused the appearance of blood in the urine of one four-year-old patient on ♏iv three times a day, which cleared up in four days after discontinuing the drug. An eight-year-old girl took iv minims three times a day for several weeks, showing no ill effects.

8. Both chronic cases give nguaiacol, ♏iv, three times a day showed marked improvement.

9. Forcing water up to 3 quarts a day of extra fluid had a beneficial effect on the case.

10. Vaccine treatment used on one chronic case shows no improvement so far.

Dr. Abt: Dr. A. K. Germann, the Senior House Physician, has studied the effect of the addition of a high percentage of granulated sugar to the food of young infants. He has made some very interesting observations and will report his results to you now.

HIGH SUGAR FEEDING

Dr. Germann: I wish to present the results of high percentage sugar feeding in 6 of our cases at Sarah Morris Hospital. Schick, of Vienna, was one of the first to try the high sugar feeding of infants. He was interested in the question of the cause of the initial loss of weight in infants during the first ten days to two weeks of life. After experimenting with a large number of cases he came to the conclusion that the initial loss was due both to lack of fluids and lack of nutrition during this period. It was in attempting to check this loss of weight that he tried the high sugar feeding. He used what he called "Kuhrog," a mixture of equal parts of cow's milk and water with cane-sugar in amount equal to $8\frac{1}{2}$ per cent. of the entire mixture. After trying "Kuhrog" on a large number of cases he came to the following conclusions:

(1) This milk mixture is harmless and is apparently relished by infants. In these respects it compares well with mother's milk and is quite superior to saccharin and tea. By giving this milk mixture directly after birth Schick found that the babies were stronger and able to nurse better when the mother's milk appeared. It may also be used as a supplement to breast milk.

(2) The initial loss in weight is much less, and in many cases there is an actual gain in weight during this period. There is rarely, if ever, a rise in temperature, and the child seems more contented and less hungry than on the breast. The meconium stool is soon replaced by a milk stool.

In short, Schick thinks that this milk mixture is well worth trying early in life, and suggests that it will work well later. His work deals almost entirely with children in the first ten days to two weeks of life. Our work here at Sarah Morris Hospital concerns children who are much older. Our youngest was sixteen days old.

Case I.—The first child I wish to present was sixteen days old, one of twins, brought to the hospital for diet regulation. The other child died at birth. The mother was attended by a midwife and developed puerperal sepsis. The feeding history shows that the child was on a milk mixture before admission to the hospital, but the amount and dilution was not known. The family history was negative save for the fact that the mother had a puerperal sepsis.

The physical examination on admission revealed a baby sixteen days old with a slight pustular eruption over the body. The temperature was normal. The urine contained a trace of albumin and many pus-cells. The blood, Von Pirquet, and throat cultures were negative.

After considering briefly the history, physical and laboratory findings, let us take up the feeding of the child during his stay in the hospital. On admission to the hospital Octover 31, 1919 the baby was put on breast milk, 2 ounces seven times a day, which was continued for four days, when another ounce was added to each feeding. Albumen-milk was substituted occasion-

ally for the breast milk. During this period of six days the child had from two to five yellow to greenish-yellow stools a day. During these six days the baby gained 3 ounces. Then we gave breast milk, 3 ounces seven times a day, with two of the breast milk feedings substituted by one-half milk containing $8\frac{1}{3}$ per cent. sugar. The next day the child was placed on the high sugar feeding without the breast milk. Note the sharp rise in weight when the high sugar feeding was added. The exact cause of this sudden rise is not definitely known, but it is thought to be due to fluid retention in the tissues brought on by the presence of sugar. The stools were quite normal and characteristic for the high sugar diet. The stools were soft, usually homogeneous, and of a greenish-yellow color, with a rather sour odor. After five days the feeding was increased to $3\frac{1}{2}$ ounces seven times a day. The baby made a rather irregular but steady gain in weight. The best continued gain was made between November 14th and December 4th—twenty days. There was a gain of 1 pound, $2\frac{3}{8}$ ounces, or 0.92 ounce per day.

On December 4th the child started to vomit and continued to do so for several days, with a fall in weight. One starvation day was followed by breast milk for several days. When vomiting finally ceased the child was again put on the high sugar feeding, and was kept on this mixture during the remainder of the stay in the hospital. The strength of the milk mixture was gradually increased.

A blood-sugar determination was made a few days before discharge and found to be 0.10 gram per cent., which is normal, showing that the sugar was probably utilized. At no time was sugar found in the urine. This baby was in the hospital for eighty-eight days and gained 2 pounds, 14 ounces—a gain of 0.54 ounce per day. During practically all of this time the child was on the high sugar feeding.

Case II.—This is an apparently normal child nineteen days old, which was brought to the Sarah Morris Hospital from the obstetrical ward of Michael Reese Hospital. The mother was unable to care for the baby. Physical examination was negative. The blood showed nothing abnormal. The urinalysis revealed

many pus-cells, which were later found to be due to a non-specific vaginitis.

On admission the child was placed on one-third milk with 4 per cent. dextrimaltose, 3 ounces seven times a day. It was kept on this mixture for seven days and gained on the average of $\frac{1}{3}$ ounce per day. It was then put on one-half cow's milk with 8 per cent. cane-sugar, 3 ounces seven times a day, which was later changed to 4 ounces six times a day. On this formula the gain was $\frac{2}{3}$ ounce per day. The stools were quite normal, with a slight tendency toward constipation the last few days. After calculating our caloric values for this high sugar mixture, we find that it is considerably above the average (45 calories per pound weight). In some cases we have given 60 to 80 calories per pound weight.

We were quite interested in the question as to whether or not the blood-sugar would be affected. A blood-sugar determination made before the high sugar feeding was instituted showed 0.127 gram per cent., while one taken after the high sugar feeding had been given one week showed 0.121 gram per cent. The sugar was probably utilized, for there was an actual decrease in the sugar content of the blood after the high sugar feeding. At no time was any sugar found in the urine. The child was discharged in good condition.

Case III.—This baby, aged two and one-half months, was placed in the hospital because of stationary weight, fever, and fretfulness. The birth was normal, and the child did well during the first two weeks of life. From that time the weight was practically stationary until the week before admission to the hospital, during which time the child is said to have lost 1 pound, 3 ounces. A week before admission the mother noted a fever, the temperature at one time reaching 104° F., and the child was fretful. The child was quite constipated at times and was given cathartics frequently. A brief review of the feeding history will explain the condition of the patient. The child was breast fed for four weeks, and was then put on equal parts of cream and water with some sugar added. This was continued until one

week before admission, when it was given two-thirds milk with 4 per cent. dextrimaltose.

Physical examination on admission revealed a thin, drawn, and markedly dehydrated infant three months of age. The anterior fontanel was markedly depressed. Save for an enlarged liver the remainder of the physical examination was negative. The urine was negative save for an occasional epithelial and pus-cell. The temperature on admission was 101.8° F., but was normal the next day and remained so during the stay in the hospital.

The feeding of this case is quite interesting. On admission the child was placed on breast milk, 2 ounces eight times a day. This was continued for four days. On the fifth day 3 ounces of breast milk were given six times a day, with one substitution of 4 ounces of one-half milk with 8 per cent. cane-sugar. The following day the baby received 3 ounces of breast milk and 20 ounces of the high sugar mixture. Schick said that breast milk and the high sugar milk could be alternated in this way. For the remainder of the stay in the hospital the child was given one-half milk with 8 per cent. cane-sugar, 4 ounces six times a day. The weight curve in this case is quite remarkable (see chart). The gain in weight on the breast milk was 1 ounce per day, while on the high sugar milk mixture it was 1½ ounces per day. I do not mean to infer that this "Kuhrog" is superior to breast milk, for breast milk surely has no superior. In all of our sick babies either breast milk or albumen-milk was given first, and the high sugar feeding was instituted only when the babies were practically normal.

Case IV.—This child was six months of age and was brought to the hospital for vomiting, anorexia, loss of weight, restlessness, cough, and recurrent sore mouth. The feeding history showed that the child had been irregularly and improperly fed. On admission the child was placed on albumen-milk and was not given the high sugar mixture until the last week of the stay in the hospital. In this case the gain in weight on the albumen-milk, with from 3 to 5 per cent. dextrimaltose, was practically the same as on the milk mixture with 8 per cent. cane-sugar. The gain

with the albumen-milk containing from 3 to 5 per cent. dextrimaltose was 0.71 ounce per day, while with the high sugar feeding it was 0.70 ounce per day. We do not use the one-half milk

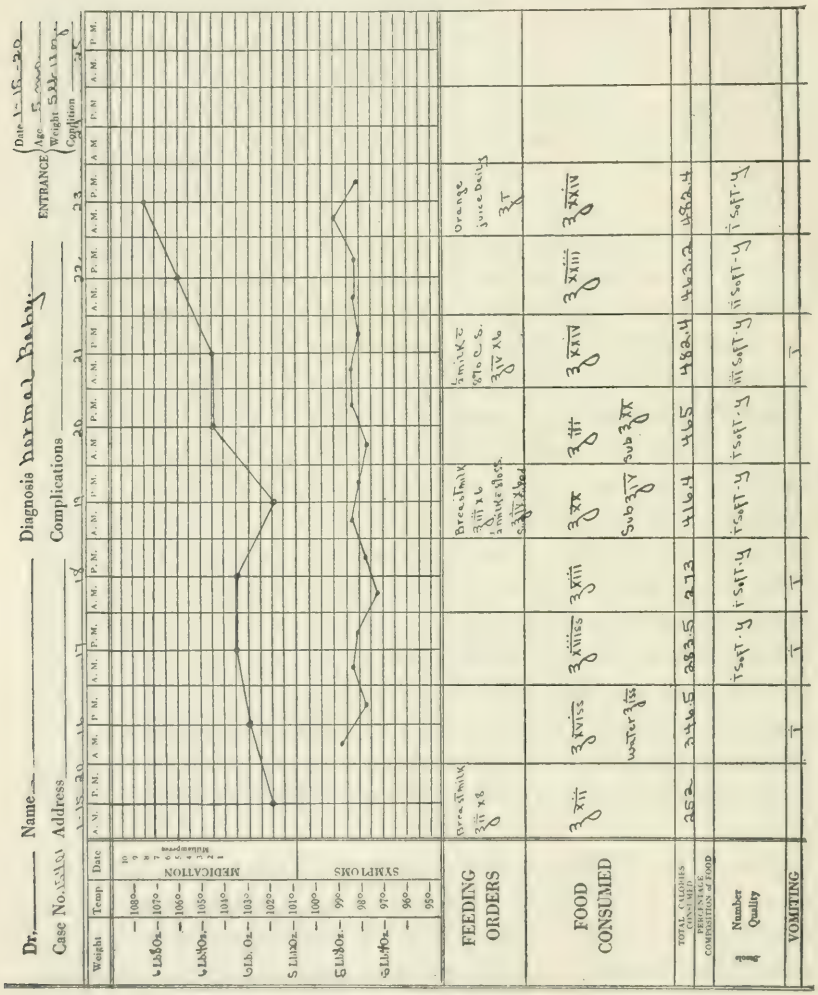

Fig. 252.

with older children, but strengthen the milk mixture in accordance with the age, always leaving the cane-sugar at 8 per cent.

Case V.—This was a male child three months of age, and was brought to the hospital for diarrhea, loss of weight, and

vomiting. The mother stated that she had been unable to regulate the diet since the child was taken from the breast at two months of age. Physical examination revealed a poorly developed and somewhat emaciated child three months of age with a slight furunculosis of the scalp. The physical examination was otherwise negative. The child was first given the albumen-milk and later the high sugar mixture. Our weight curve shows that the gain in weight on the 8 per cent. cane-sugar mixture was 0.09 ounce per day, while on the other mixtures there was a gain of 0.63 ounce per day. The stools were quite normal. The furuncles on the scalp were slightly more marked on the high sugar feeding. We find that any infection greatly affects the weight curve of the baby.

Case VI.—This was a female child five months of age, who was brought to the hospital for care while the mother was undergoing an operation. Physical examination revealed a normal, well-developed, and well-nourished child with a slight eczema of the face and chest. The child developed a mild pyelitis and otitis media during her stay in the hospital. On admission the child was placed on albumen-milk for a few days, but was soon given a milk mixture with 4 per cent. dextrimaltose. This was continued for two weeks, when the high sugar feeding (8 per cent. cane-sugar) was instituted. The child was also given farina paste and orange-juice. Our result here is quite remarkable. On the 8 per cent. cane-sugar mixture the child gained 0.67 ounce per day, while on the other feeding there was a slight loss of 0.005 ounce per day. The high sugar feeding, moreover, was given during an attack of otitis media. The child was discharged in good condition, the eczema having disappeared.

Our tabulated results are as follows:

Case I.—Gain in weight per day on the 8 per cent. cane-sugar mixture was 0.54 ounce per day.

Case II.—Gain on the 8 per cent. cane-sugar mixture was 0.67 ounce per day. Gain on the other mixtures was 0.34 ounce per day.

Case III.—Gain on the 8 per cent. cane-sugar mixture was 1.50 ounce per day. Gain on breast milk was 1 ounce per day.

Case IV.—Gain on the 8 per cent. cane-sugar mixture was 0.70 ounce per day. Gain on the other mixtures was 0.71 ounce per day.

Case V.—Gain on the 8 per cent. cane-sugar mixture was 0.09 ounce per day. Gain on the other mixtures was 0.63 ounce per day.

Case VI.—Gain on the 8 per cent. cane-sugar mixture was 0.67 ounce per day. Loss on the other mixtures was 0.005 ounce per day.

In summarizing the findings of our cases we conclude that the 8 per cent. cane-sugar mixture certainly does no harm, and is apparently relished by infants. When given alternately with breast milk the infants do very well. In the majority of our cases the gain in weight was greater with the high sugar feeding than with other mixtures.

DR. ABT: This concludes this morning's program. I am very glad to have had the opportunity of letting these younger men tell you for themselves about the clinical studies that they are making, and I hope it will be possible from time to time for them to present further reports to you. I know from the enthusiasm that you have shown this morning that you will await them with interest.

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

LYMPHOSARCOMA OF THE NECK

February 26, 1920.

THE patient whom I wish to show you today is a colored laborer thirty-seven years of age, who presents the following history:

Complaints.—He complains, first, of swelling in the neck; second, of very great swelling of the face, which came on subsequently to the swelling of the neck; third, of "lumps" in both axillary regions, and, lastly, of some difficulty in swallowing and speaking.

Previous History.—The patient states that in 1914 he had a severe neuralgia of the left side of the face. This involved particularly, indeed almost exclusively, the region of the gums, and at this time the gums burst open and discharged pus on three separate occasions, the last of these being October 15, 1919. The amount of pus which came out on each occasion was quite considerable and the patient refers all of his trouble to this. On October 16, 1919, the day after the last abscess in the gum ruptured, the face began to swell, so that it was very painful. On the following day the patient went to a physician, who discovered an enlargement of the lymph-glands or, as he says, "knots," on the left side of the neck.

Questioning the patient a little more closely we learn that he felt pretty bad for a month or more before visiting the physician, that he had been weak, lacked "pep," and felt rather wretched.

Swelling of the Neck.—When we inquire about the swelling of the neck more particularly, we find that it has increased with

great rapidity. He states emphatically that when the doctor found these "knots" on October last they were so small that he himself did not believe the doctor was correct. The physician told him at that time that they were the size of small hickory nuts. When asked in which particular region the glands were located he points to the anterior and posterior triangles of the neck. His physician told him that his teeth were at fault, and so on December 31 1919 he had all of his teeth extracted. He

Fig. 253.

had a great deal of pain following this extraction, and the glands increased in size very rapidly on both sides of the neck. I believe I stated that the original enlargement was on the left side, but the right side of the neck developed knots of sufficient size for him to see and feel by the latter part of October or the first of November. All of these enlargements were painless, except that one gland impinged upon the left ear and produced an earache. None of the glands were tender to the touch.

Swelling of the face has been present for the last three weeks, gradually increasing, so that at the present moment both eyes are almost closed. The swelling began about the ears, first on the left side, and has increased so that it is generalized. It is soft and typically edematous.

Axillary Enlargement.—The patient noticed the axillary enlargements for the first time on February 23d of this year.

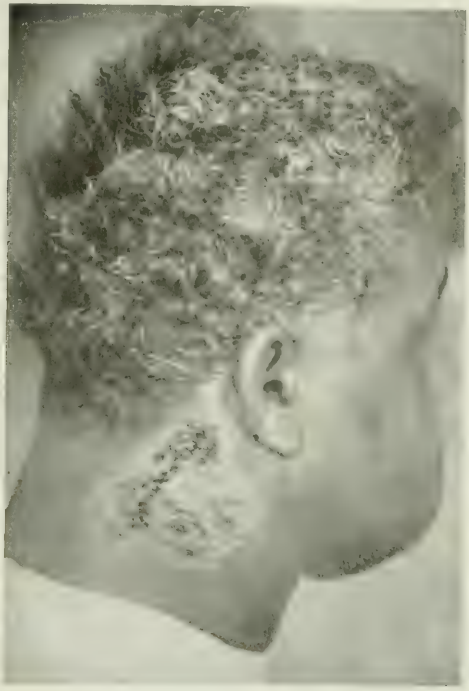

Fig. 254.

He noticed then that lumps as big as hickory nuts were present in both axillæ. He states that he had a "pimple" in the right axilla which caused considerable soreness, and on investigating the pimple he noticed the axillary enlargement. So far as he knows he has not, and has not had, any other swelling or enlargement.

Difficulty in swallowing has been present during the past

week only. The patient noticed the pressure on his neck, and says that it has frequently produced some regurgitation. The food seems to go down a very little way and then comes back, without any nausea.

Fig. 255.

Difficulty in speaking has been present only during the past week, and for the most part of this time he had not been able to speak above a whisper.

The patient states that he has no other symptoms than those enumerated.

Inventory of General Condition.—Summing up the history of the patient as he gives it to us, we see that the symptoms are very evident and the cause for them, of course, lies in the enormous glandular enlargement which you see at a glance on either side of the patient's neck. He had lost about 15 pounds since December, but thinks he has regained some of this. He had had only a slight cough, no expectoration, no dyspnea. His appetite is good and his bowels normal. No abnormalities are present in the genito-urinary system, in particular, no hematuria.

I think you will all be impressed by the fact that the patient's answers are intelligent, that he seems alert, and is quick to understand what we desire of him. I have myself questioned him very carefully and feel sure that the history is much more accurate than we ordinarily obtain from patients in his social stratum.

Previous Illnesses.—We learn that he had typhoid as a child, mumps at sixteen years of age, measles and a left-sided pneumonia some years ago; many recurrent attacks of tonsillitis when he was about seventeen. He still has his tonsils, although he has been advised to have them removed. He has never had scarlet fever or diphtheria, and no glandular enlargements in other parts of the body. Some years ago he had a cut on the middle finger of his left hand from which he had "blood poisoning," which was quite severe.

Family History.—This is substantially negative. One brother died of pulmonary tuberculosis, but we can find no evidence of glandular enlargements of any sort in the parents.

Venereal History.—The patient denies lues, but had a urethritis eighteen years ago and has had many recurrences of this from time to time. He has at present a well-defined stricture of large caliber.

Habits.—He uses alcohol to a limited extent and smokes in moderation.

Physical Examination.—The patient at first sight looks fairly well nourished, but I think you will agree with me in saying that

he is much more emaciated on the lower part of his body than he appears to be above the waist, more particularly above the nipple region. This is due to the edema which is so high grade that it gives an appearance of his being unusually stout. The legs and thighs, however, tell the tale that he is in reality rather emaciated than otherwise.

Head.—The scalp is negative; the eyes are almost closed from the edema of the lids, but otherwise show no abnormality. The face, nose, and ears show nothing aside from edema. The mouth shows only one remaining tooth and the gums and buccal mucosa are very edematous. The tongue is swollen to at least twice its normal thickness, the swelling apparently being due to edema. The posterior wall of the pharynx is edematous and pale and shows a distinct forward bulging.

Neck.—We can distinguish plainly two varieties of enlargement: first, a diffuse enlargement due in part to edema which is exceedingly hard. One can make an impression with the finger, but the neck is of almost leathery consistency. The circumference about the middle of the neck is 51 cm. The cervical lymph-glands, anteriorly and posteriorly, superficial and deep, are all very greatly enlarged, apparently immovable, and matted together. It is almost impossible to move the skin and subcutaneous tissue over the tumor masses, so tightly is the skin bound down to them. On the left posterior aspect of the neck there is an ulcerating area about the size of a quarter, which is exuding a small amount of bloody pus. The edges of the ulcer, which is quite superficial, are ragged. The enlarged glands can be traced down into the cavity of the chest with great ease, particularly into the anterior and superior mediastinum. They encroach upon the ears and extend practically from the midline anteriorly to the midline posteriorly.

Thorax.—The edema and induration of the tissues extend down over the entire surface of the upper sternum, reaching practically to the nipples, and posteriorly extend down over the scapulæ into the interscapular space. The edema becomes less as it extends down and is not nearly so marked as in the neck

region. It seems much fresher in that we can pit it very much more easily than is the case in the neck.

The *heart* is substantially negative, the upper boundary being at the lower margin of the third rib and the apex-beat in the fifth intercostal space, well inside the nipple. There are no abnormal thrills or murmurs. The transverse dulness over the upper sternum is greatly increased and amounts almost to a definite flatness.

In the *lungs* there is impaired resonance over both apices and above the scapulæ posteriorly. The breath sounds are distant over the above areas. No râles can be heard at the present time, and the voice sounds seem to be normal. In particular, we can find no evidence of fluid in the pleura.

The Abdomen.—The liver and kidneys are not palpable; the liver dulness is of normal extent. Placing the patient in the supine position we are unable to palpate the spleen, although his abdomen is soft and relaxed, and we can find no increase in the splenic dulness. No evidence of fluid can be found even on placing the patient in the knee-chest position.

The *genitalia* are negative, except for the stricture above referred to.

The Extremities.—The upper extremities show a flexion contraction of the middle finger of the left hand. The axillary glands on the left side are enlarged to the size of walnuts. They are distinctly discrete, quite hard, and freely movable under the overlying skin. The right axillary glands are considerably larger and fused together into a mass of considerable size. They are not tender, not fluctuating, and not adherent to the overlying skin. The epitrochlear lymph-glands are just palpable.

The lower extremities are negative except for several copper-colored scars along the anterior surface of both tibiæ. The inguinal glands are enlarged to the size of beans, discrete, not tender or painful.

The Reflexes.—The deep reflexes are all present with the exception of the knee-jerk, which is absent on the left side and sluggish on the right.

Blood-pressure.—The blood-pressure is 140 systolic, 84 diastolic, pulse pressure 56. The Wassermann reaction on the blood is frankly negative.

Summing up the case as far as we have gone the findings are relatively simple. A man who has had much trouble with his gums and teeth commenced to feel weak and run down, and after a few weeks develops a cervical glandular enlargement present on both sides, which grows with great rapidity, so that from Octover 15th to the present, approximately four months, we have these quite large tumor masses. Referring to the patient's statement that the glandular enlargement followed immediately upon his last large abscess in the gum, let us examine the neck again carefully and see if this had any causal relationship.

On careful palpation we do not find any enlargement of the submaxillary and sublingual glands, and inasmuch as he had had several previous abscesses in the gum I think we can at once rule out any suspicion that these are simply local glandular enlargements following upon a severe septic condition in the mouth. This idea is further borne out by the fact that removal of these teeth, with the exception of one, had no effect upon the glandular enlargements except, apparently, to stimulate their growth.

I hardly need say to you that there is scarcely any condition in which our knowledge is in such a chaotic state as it is in regard to the diseases of the lymphatic apparatus. Different authors use the same terms in entirely different senses, and what one author means by a "lymphatosis" or a "pseudoleukemia" is entirely different from that another author of equal experience may mean. I do not wish to enter upon a discussion of the pathology of the diseases of the lymphatic system because the time is hardly ripe when one may do this with profit, but we must go into them at least sufficiently to see whether we can arrive at a definite diagnosis in this case.

To begin with, in all sorts of septic processes about the mouth the experienced clinician will at once think of the acute leukemias. I have seen quite a number of these, and in three or four at least

the leukemic condition was entirely overlooked. The condition in one instance was supposed to be a local disease, confined to the mouth and to the tongue. In another patient, with acute leukemia, the disease appeared limited to the tonsils. In a third patient there was a general septic condition, a little like the one before us, in addition to a very severe ulcerative stomatitis. Taking another careful look at his throat, we see only that which the history states, the tonsils are moderately enlarged and show evidence of old chronic inflammatory processes, but nothing in any way comparable with the glandular enlargements in the neck.

Our first task, then, I should say would be to rule out the acute leukemias. We would, in all probability, have in these an enlargement of the spleen, and this, as I have already stated, is entirely lacking. The blood examination must decide the matter. We find the patient has 4,600,000 reds, 12,500 whites. The differential count shows 15 per cent. small mononuclears, 16 per cent. large mononuclears, 68 per cent. polymorphonuclear neutrophils, and 1 per cent. eosinophils. No abnormal cells were noted. Let us ask ourselves whether this is the picture of acute leukemia. I think we must answer very definitely "No," although we must not forget that we do have aleukemic intervals in some of these cases which are exceedingly puzzling. I have seen one or two such cases myself, but I cannot conceive of a glandular enlargement such as this with the blood findings just enumerated. I think, therefore, that we can eliminate acute leukemia or, for that matter, leukemia of any type.

Bearing in mind the frequency of tuberculosis in the colored race leads us to think of the possibility of these being ordinary tubercular glands of an unusually acute type. The differential points are not so easy to state with exactness. However, we must consider the following: Tubercular glands almost never grow with such rapidity as these. It would be exceedingly unlikely that they would attain such a size and become so matted together without the usual evidences of softening and without considerable pain. In addition, we would probably expect considerable fever, and up to the present time the patient has

shown but very little rise in temperature, it not having been above 99° F. in the two or three days he has been in the hospital. The probability is very strongly against this being tubercular in nature.

Let us examine the ulcer referred to in the side of the neck and see if we can get any information from it. It seems superficial and the patient himself tells us that it came on after putting a very hot water-bag to his neck. The appearance of the ulcer is entirely compatible with such an explanation. However, we have had scrapings made from the surface of the ulcer and examined with particular reference to actinomycosis and similar organisms, but with negative results, nothing but a few pus- and blood-cells being found. I do not believe that the ulcer is due to the glands themselves coming through the skin, but am inclined to think the patient's explanation is correct.

We might think of syphilitic glands, but the mode of their development and the absence of a positive blood Wassermann reaction both speak against this. I have never seen syphilitic glands of such size and with the tendency to mat together that these show.

By far the most difficult point in the diagnosis is to settle whether or not this is a Hodgkin's disease, using the term "Hodgkin's disease" in the restricted sense in which it is now used by our best recent authors. Hodgkin's disease is common enough at this man's time of life. It begins as, apparently, this case did, in the cervical glands or in the upper mediastinal glands. The glandular enlargements are quite frequently as pronounced in Hodgkin's disease, indeed, frequently more pronounced than we have in this case. On the other hand, Hodgkin's is rarely so acute. In a good many cases I do not think I have ever seen one which produced such sizable masses in the short period of four months. We may, however, be deceiving ourselves in one respect, namely, in that these cervical glands may be only the outposts of much larger masses of mediastinal glands. We have already noted that there is a marked dulness across the upper sternum, indicating that the superior mediastinum contains glandular enlargements. We have here an *x*-ray plate of the

chest and, as you see, it shows nothing except that the superior mediastinum shows an increased density and there is a slight density at the top of each lung, particularly on the left side. The hilum shadows are usually well marked. It also confirms our physical findings in that we find no evidence of metastases in the lungs or fluid in the pleura or pericardial cavities.

The blood examination is certainly not decisive one way or the other. My own judgment is that it is certainly very exceptional that we can make a diagnosis of Hodgkin's disease from the blood. I have never seen a case in which I thought this could be done with any degree of certainty. We may say, then, that the evidence against Hodgkin's disease resolves itself into the fact that the glands have grown with greater rapidity than we would ordinarily expect.

We now come to what seems to me to be the *real, decisive, differential point, and that is the fact that the glands have infiltrated the surrounding tissues.* That is not ordinarily the case in a true Hodgkin's disease and, according to my experience, removes the case at once from that category and places it in the group of the malignant or, at least, semimalignant tumors of the lymphatic glands; in other words, to the group of the lymphosarcomata. We may distinguish in general two groups of the lymphosarcomata; a slow-growing and a rapidly growing group. The glands in the rapidly growing variety develop in a few months, as these have done, become matted together, *and after a while infiltrate the surrounding tissues,* producing great pressure on the surrounding organs and causing secondarily such symptoms as this patient shows, namely, edema, difficulty in swallowing and speaking.

The blood examination fits in perfectly well with the diagnosis of lymphosarcoma, although there is nothing absolutely characteristic about it. The only safe way to deal with a case of this sort is to remove a small gland and submit it to microscopic examination. In the short time which the patient has been in the hospital we have not yet had time to do this, but will do so now and report to you at the next clinic, a week from today.

March 3, 1920.

You will remember this patient whom I showed you last week and on whom I made the diagnosis of lymphosarcoma of the cervical glands. After the clinic we removed one of the axillary glands and we now have the report from Dr. Stangl as to its nature. I will read it to you verbatim: "*Histologic diagnosis:* Microscopic preparations of these lymph-glands present characteristics of lymphosarcoma. Many mitotic figures are present."

I have very little further to say about the case. The diagnosis being thus verified, the question is, What shall we do with the patient? Extirpation of these glands is, of course, impossible, and the thing which seems to promise the most benefit is intensive *x*-ray treatment. I have already instituted this, and you will see that even after so short a period of treatment the glands look distinctly smaller than they were when I showed him to you last week. We shall continue this treatment and I will show him to you again at our next clinic. It not infrequently happens that the glands in these patients undergo very rapid diminution without, however, materially changing the ultimate prognosis.

March 10, 1920.

Just a few words more in regard to the patient with the lymphosarcoma. Under the *x*-ray treatment the glands diminished greatly in size, so that the edema of the face entirely disappeared and one could see very plainly that the man was, in reality, greatly emaciated. Just after I last showed him to you the patient's temperature arose suddenly to 104° F. and for the few days following fluctuated between 101° and 104° F. The cause of this we soon discovered to be a right-sided pleuritis, which was shortly followed by a left-sided involvement. The day before the patient's death, which occurred six days after I last showed him to you, he commenced to complain of pain in the abdomen, but we could not, because of his desperately sick condition, make very much out of it. We regarded the pleuritis as a terminal infection, probably of pneumococcic or streptococcic origin. Coincident with the rise in temperature the white

blood count rose to 26,000 and the polymorphonuclear neutrophils to 90 per cent. of the total count.

An autopsy was obtained, and the findings, in brief, were these: Primary lymphosarcoma of the mediastinal, cervical, and axillary lymph-glands. Bilateral serofibrinous pleuritis; fibrinous pericarditis and peritonitis. There was a small healed focus of tuberculosis at the right apex.

The most interesting feature to me in the autopsy was the examination of the cervical glands which had been subjected to *x*-ray treatment. The glands in the more superficial portions were atrophic, much more fibrous than ordinary, and cut with increased resistance. These findings were in striking contrast to the deeper glands of the cervical region, which did not seem to be different from normal. There has not been time as yet to study the glands histologically, but it was sufficiently obvious from even the macroscopic examination that there had been a very material change brought about by the application of the *x*-ray. This, of course, is not new, but it is interesting to see that, so far as we could judge, only the superficial glands had been materially affected.

It is a question which might be debated as to whether the glandular enlargement began in the cervical or anterior mediastinal glands. Dr. Stangl regarded the mediastinum as the most likely origin. It is instructive to know that the pleuritis was not due to the presence of tumor metastases, but rather to a terminal infection, as we had assumed. These terminal infections are, of course, so common that we hardly need to discuss them. The exudate on the surface of the pericardium was quite fresh, and as the man's condition during the last forty-eight hours of his life precluded examination, this had escaped observation, as it generally does in such cases.

PERNICIOUS ANEMIA WITH EXTREME DROPSY. A RECURRENCE AFTER A FIVE-YEAR INTERVAL

March 15, 1920.

THIS patient is an Italian laborer thirty-three years of age, who came into the hospital with the examining room diagnosis of nephritis. His present complaints are swelling of the feet and shortness of breath. He says that this condition started about thirteen months ago with a cold. A short time after the cold he had shortness of breath and pain in the stomach. He began to be "yellow" five months ago. He had nosebleed five weeks ago and following this the legs and body began to swell. The legs were first affected, then the abdomen, and then the upper portion of his body. He had nosebleed about four times in two weeks, bleeding pretty freely each time. He was in another hospital for four months. Subsequent inquiry developed the fact that he had an attack similar to this five years ago and was sick for sixteen months. At this time he was "yellow," too, but the yellow color disappeared when he got better. He had no pain in the abdomen. I have gone into this rather carefully, and find that five years ago he became sick, his feet and ankles swelled, and this condition lasted for nearly sixteen months, and then cleared up completely, and he was well until his present symptoms started thirteen months ago. All this time he did laborious manual work with ease. So we can look upon this as his second attack of very severe edema.

Pathologic History.—He has always been well except for the usual diseases of childhood. Has never had rheumatism, tonsillitis, or sore throat.

Habits.—He has drunk a little whisky and beer all his life, but says he is more temperate than most men. His bowels are always normal. He gets up occasionally at night to urinate.

Family History.—Mother died at thirty-four in childbirth. His father is living at seventy-three. A venereal history is denied. He has never been married.

Without reading the history of his physical examination when he came in, we will look at him and see what we find at present, because his condition is not materially different from what it was when he entered the hospital on the 10th of this month. You will see that he is swollen almost beyond recognition as a human being. He has, perhaps, as high a grade of edema as one will ever see. The calf of the leg can be pitted to 1 or 1½ inches. The thighs are enormously edematous and the edema is not quite fresh, since it is no longer perfectly soft. The belly is distended almost like that of a pregnant woman. The abdominal wall is markedly edematous, and without going too much into the details I will say that he has fluid in the abdominal cavity, although not as much as one might think, since much of the fluid is in the abdominal wall. That portion of the body above the nipples is fairly free from edema in comparison with the rest of his body and yet shows quite a good deal. The patient says he has been jaundiced right along, but is getting more jaundiced all the time, but when we look at him closely we will see that he is really pale, and while he has a yellow tint, it is not the tint of a jaundice. The ocular conjunctiva and conjunctiva bulbæ are both very pale, and there is not the slightest trace of real icterus present.

The urine has been examined and we find no trace of bile-pigment in it. The urine shows nothing special, but he is passing a little bit more than ordinary at present because of diuretics. The specific gravity runs 1012 to 1015, no albumin, no sugar, no formed elements except an occasional hyaline cast. The blood-pressure is low, systolic 110, diastolic 58.

Physical Examination.—Examination of the various organs shows the following:

Head.—Eyes, ears, nose, and throat are all perfectly normal except that the teeth are in very bad order, to which I call your particular attention. The lips are very pale and I want you to notice particularly his tongue. It is very pale and it is also very smooth. That is one of the atrophic conditions of the tongue similar to that seen occasionally in syphilis. The significance is not specific; there is nothing about the tongue that you

can diagnose from the tongue itself, but it looks as smooth as glass.

Lungs.—There is nothing special about the lungs except that there are a few râles at the base posteriorly.

Heart.—The outlines are very difficult to make out. The abdomen is so distended that we expect the diaphragm to be pushed up, and so we are not surprised when we find the apex-beat just about in the nipple line, behind the fifth rib, and in the fifth interspace. In the second interspace on the left we hear a loud, harsh murmur with a slight accentuation of the second pulmonic.

Abdomen.—We cannot tell anything about the condition of the abdominal organs; the abdomen is so full of fluid that nothing can be made out with certainty.

The *fluoroscopic examination* shows a very greatly increased heart shadow, especially toward the left, and the suggestion made by the roentgenologist is that the character of the shadow suggests the possibility of a pericardial effusion.

Temperature.—There has been very little rise in temperature; it has not exceeded 99° F. and a fraction since his entrance.

The feces have been examined carefully and there is nothing abnormal about them, except a moderate number of Trichomonas intestinalis. The Weber test is negative for occult blood.

At first sight this case does not look to be an unusual one. It looks just like one of the common cardiovascular cases that most of us might pass by and think it was just an old broken-down, decompensated heart. What do you think is the probable diagnosis?

VISITING PHYSICIAN: It might be some renal trouble, although the urinary examination does not show it. It might be either an acute or chronic nephritis with secondary cardiac breakdown.

ANOTHER PHYSICIAN: I was thinking of a malarial cachexia.

DR. WILLIAMSON: That would not explain his cardiac findings, edema, etc.

VISITING PHYSICIAN: I thought it might be cirrhosis of the liver or cardiac trouble.

DR. WILLIAMSON: Those were the things that first came into my mind, but they are easily disposed of. It is not a chronic interstitial nephritis, because if it were we would have a high blood-pressure instead of the low pressure. Neither is it an acute nephritis. The urinary findings are absolutely negative as regards both these diseases.

Two things speak strongly against a portal cirrhosis. In the first place, we have the history of the onset of the edema being in the legs and the history of the shortness of breath and so-called "jaundice" which lasted for many months before the edema. Then we have the history of his having had this some years ago for a period of several months, of its clearing up, and now returning. In a portal cirrhosis the fluid does not go away for years and then come back. Then, again, as we look at the patient now we find that a large portion of the fluid is in the abdominal wall and not in the abdominal cavity.

Now as to the *cardiac* conditions: Of course, it is extremely difficult, under these circumstances, to determine the exact location and size of the heart, so our physical findings are likely to be at considerable variance with the facts. We find the apex-beat pushed a little up and out, being in the fifth space and behind the fifth rib in the nipple line, and this suggests a pushing up of the diaphragm. Without knowing anything about the heart the roentgenologist suggests the probability of pericarditis with effusion. In regard to the murmurs in the heart, the only thing we can find which is at all constant is the loud systolic murmur over the base, and that is characteristically accidental. It is a loud, harsh murmur heard almost exclusively at the base, and which varies greatly in its intensity from day to day. I have half a dozen patients in private practice who have exactly the same murmur. The precise significance of these murmurs is still a matter of discussion.

Now what would be present if this were a pure heart lesion? Where does a broken compensation first show itself? In the liver. So, even if we can't find any enlargement of the liver, it certainly ought to be very tender. The higher up we go in the abdomen, the less edema there is. I can't feel the liver at

all, but when we percuss we get the normal liver dulness. I think we may safely say that this man has no renal condition, no primary heart lesion, and no cirrhosis. But when we have eliminated these three things we have eliminated the three most common things which produce dropsy. What could he have which would produce his present condition?

The answer to that question is found in the examination of the blood. The first examination showed a red count of 719,000; there were 8000 white corpuscles, with 23 per cent. hemoglobin, giving a color-index of 1.6. That was when he came into the hospital four or five days ago. Just before coming into the clinic I had another count made and it checks up substantially. This time there were 800,000 reds and 8000 whites. The differential count showed 54 per cent. polymorphonuclears, 2 per cent. large mononuclears, 40 per cent. small mononuclears, 4 eosinophils, no basophils, no myelocytes. Macrocytes, microcytes, poikilocytes, and polychromes were plentiful. There were some small nucleated reds, some normoblasts, and a number of megaloblasts. We have a stained specimen of his blood under the microscope, and you can see as pretty a picture of pernicious anemia as you have ever seen.

The interesting thing about the case is that the symptoms of pernicious anemia are so beautifully masked under the heart condition. His condition is typical of heart disease if it were not for the fact that his previous condition lasted for sixteen months and then cleared up and remained well for *five years*, until he developed the present condition. A pure heart condition does not often, if ever, last for sixteen months and then clear up without showing some dyspnea, especially on exertion, in the intervening time.

The pernicious anemia explains another condition. When the man came into the hospital he said his doctor told him he was "jaundiced" and that he had been for a long time. But, as you see, he has no jaundice at all. His color is the typical lemon yellow of pernicious anemia and there is no bile-pigment at all in the urine. We would like to know if the spleen is enlarged, but it is impossible to determine this with certainty.

I have seen a great many cases of edema, but have never seen a much higher degree than this. Patients with pernicious anemia not infrequently develop an edema toward the end, but this is extraordinary. We may picture to ourselves that the pernicious anemia has so affected the heart muscle that it has become dilated and incompetent, just as in an ordinary myocardial degeneration. But sometimes we find edema without the enlargement and tenderness of the liver which we are accustomed to look for in cases of myocardial insufficiency. The precise *modus operandi* in which the anemia produces the dropsy is unknown, but it occurs in a very fair number of cases of pernicious anemia. It would, *a priori*, seem quite probable that in just the same way that it produces a myocardial degeneration it may produce a degeneration of the capillaries, and this, in turn, cause the edema.

There is one other small point which may possibly be interpreted in this sense, and that is the repeated attacks of epistaxis which the patient has had. While small hemorrhages, especially retinal hemorrhages, are not very rare in pernicious anemia, really sizable hemorrhages are distinctly uncommon. Their presence in this case points, I think, to impaired vitality of the blood-vessels. The epistaxis did not amount to but an ounce or two each time, so that it was entirely inconsequential in the production of the anemia. Furthermore, the blood-picture is not at all that of a secondary anemia.

How about his previous condition? Was that pernicious anemia? We know that a great many, perhaps most, cases of pernicious anemia have at least one remission. You will often find that a patient who gets very low in his blood count will sometimes go back very nearly or quite to normal. A very interesting case came into the hospital with a diagnosis of pernicious anemia a number of years ago, and he rapidly got so much better that they were convinced they had made an error. The patient went out and stayed for some time and then came in again in just the same condition. This was four or five years later. Again he improved very markedly and went out, but came back seven years later with another attack, and the autopsy

showed typical pernicious anemia. So you can see what great remissions these patients sometimes have.

I regard the history in this case as the strongest point in the diagnosis outside of the examination of the blood, *i. e.*, the fact that five years ago he had the same shortness of breath and the same swelling and yellow color which lasted sixteen months and then cleared up entirely, the man staying well for five years and doing hard manual labor. This attack is certainly an extreme one. The entire blood-picture, the differential count, the low white count, the extraordinarily low red count, the high color-index, the presence of macrocytes, and nucleated reds and tremendous poikilocytosis point with certainty almost toward a pernicious anemia. Even if there should be found an organic heart condition my judgment as to the pernicious anemia would not be changed.

One must be very careful in the judgment of the results of treatment of pernicious anemia. Everybody knows that in cases of pernicious anemia when the patient has a remission they are sure that whatever they have been doing for this patient has caused the remission. We have seen that many times. Suppose I had given this patient a blood transfusion five years ago. I could have showed him to you a year or two ago as an example of what it had done. We must be very careful in our estimate of therapeutic results, because if we are not, we will make all sorts of mistakes.

Note.—The patient died a few days later.

Autopsy Findings.—The findings may be summarized in a few words by saying that aside from a high-grade fatty degeneration of the myocardium the heart was negative. The kidneys showed only fatty changes. Otherwise the findings were typical of pernicious anemia. The spleen was moderately enlarged, a condition which we had not been able to make out during life.

CLINIC OF DR. SOLOMON STROUSE

MICHAEL REESE HOSPITAL

URTICARIA AND ANGIONEUROTIC EDEMA

Illustrative Cases; Full Discussion of Etiology; Treatment and Management

TODAY I wish to present to you some cases of two conditions, which, if they are not manifestations of the same thing, are at least so closely related as to warrant inclusion under the same heading. Urticaria and angioneurotic edema are common enough, yet very little is understood either about their fundamental etiology or their treatment. In reviewing my records I find several patients who have had both urticaria and angioneurotic edema, occasionally synchronously and at other times one or the other. In many ways the conditions seem to resemble one another and, perhaps, both are only different manifestations of the same erythema group of skin diseases, concerning which Osler has written so fascinatingly and instructively. It has seemed almost impossible to make an orderly presentation of this subject, so I shall merely present a few cases, each one of which emphasizes some special feature on which to hang discussion.

CASE I.—S. L. S. Urticaria. This man was first seen by me in November, 1912, at which time he was fifty-six years old, a paint manufacturer, and married. He complained of attacks of hives almost every night for the past two years. In his family history are relatives with hay-fever, diabetes, gout, migraine, and excessive adiposity. His own past history is practically negative. His habits are excellent, does not smoke, occasionally took alcohol, plays a good deal of golf, and is admittedly of a neurotic temperament. In October, 1910, one evening after a meal containing grapes and beer the patient

was suddenly seized with an attack of hives and "he has never been without them since." Patient described his eruption as being typically urticarial in character, which attacked almost all parts of the body. Occasionally there have been swellings of the eyelids and lips; attacks usually appeared every evening. However, the patient volunteered the information that during a card game or any other occupation which took his attention from himself he did not notice the rash, even though it was present. During the two years before I saw the patient he had been under treatment by various men. He has always been greatly constipated, but otherwise has no gastro-intestinal symptoms. Examination showed a healthy, robust man with a normal cardiovascular and urinary system. The skin was extremely sensitive to any irritation, factitious dermographia being produced by the slightest irritation. His blood count showed: erythrocytes 4,864,000, leukocytes 12,400, hemoglobin 90 per cent.; differential, polymorphonuclears 78, large lymphocytes 5, small lymphocytes 17, eosinophils none, basophils none. The urine showed an excess of indican, otherwise negative. The Ewald meal showed no free hydrochloric acid, total acidity of 17.5, no lactic acid, no blood. The feces were large, constipated, brown, no gross abnormalities, and microscopically showed an excess of fatty acids and soaps. Red blood-cells were present and both benzidin and guaiac tests were positive. The presence of blood, however, could be accounted for by hemorrhoids.

In the patient's history was evidence of periods of remission when the patient's diet was entirely free of animal protein, but the urticaria immediately returned on resumption of protein diet. It will be noted that the blood-picture was that of a slight infection as indicated by the absolute increase in leukocytes, absolute increase in polymorphonuclear neutrophils, and complete absence of eosinophils. There was likewise present a long-standing history of constipation, with an increased indican reaction in the urine.

We then reasoned that the patient's urticaria was a nonspecific protein intoxication associated with intestinal stasis

and some localized infection. Since the physical examination had not shown any evidence of infection elsewhere in the body, it was thought that the infection might be located in the intestines, and that as a result of the combination of infection and stasis protein was broken down into substances toxic to this individual. The temporary symptomatic relief which had followed dietary restrictions would be explained by the removal of the protein, whereas the recrudescence of symptoms upon a return to normal diet was explainable by the failure to remove the primary cause of the trouble.

If this conception were correct, therapy should aim, first, to remove protein; second, to relieve the stasis, and third, to change the intestinal flora. The work lasted almost four months. Treatment in brief was as near a protein-free diet as possible, the feeding of Bulgarian bacilli, and the injection of the posterior lobe of pituitary extract. After a far from uneventful course the patient on March 1st was on a practically normal diet, but was free from hives. During the past seven years he has remained free.

Case II.—C. H. S. The second patient is a young woman thirty-five years old, married, whom I first saw in 1916 for hives. She had been under treatment at that time already for some months. She could give no facts in her history regarding any possible cause or relationship between the attacks of hives, food, or other things in her daily life. She was a woman of usually good health with no particular facts in her past history. The family history was characterized by the fact that almost all members were neurotic. The treatment to which she had been subjected was mainly dietetic and hygienic, but nothing seemed to have helped her, and she was constantly losing weight and constantly having hives. The examination showed a small woman, skin was very dry, eyes slightly prominent, considerable loss of weight, the general examination negative. The urine showed nothing abnormal. Sodium chlorid and urea excretion were normal. No indican was present. The blood examination was normal, no eosinophilia, hemoglobin 85 per cent. The

patient suffered from diarrhea and the stool was large, semisolid, greenish-brown in color, odor very offensive; microscopically, much undigested food, especially the fibers of vegetables and fruit, and some mucus was seen. During the time this stool was examined the patient was on a vegetarian régime and, as just stated, suffered from diarrhea.

After many attempts at trying to establish a dietary etiology, the patient's loss in weight and strength became pronounced, she was likewise quite nervous and worked up about herself. Finally, her husband and I agreed that we were doing more harm than good, and decided to allow her a free diet, assuring her at the same time that we had been convinced food had nothing whatever to do with her urticaria, and that if she built up her strength her hives would disappear. Incidentally, some time before we had done this we had removed some blood and reinjected the serum, which you may recall was being advocated at that time as a cure.

The surprising feature of this case was that as soon as we stopped treating the patient she got well.

Case III.—B. W. Male, white, single, nineteen years old, salesman. Angioneurotic edema. Seen in April, 1919. Eight years ago following an attack of scarlet fever this young man noticed that suddenly on different parts of his body "swollen areas" appeared and disappeared within twenty-four hours. About a year ago he had a recurrence. The patient noticed that for weeks at a time he would have no attacks, and then, like a bolt from the blue, "the thing would appear and disappear as quickly as it came." For the past year he has been constipated. There is no cough, no sore throat. He eats everything. "Bled freely after the extraction of a tooth some months ago." It is impossible to get any family history from this individual. Examination showed many interesting findings, so it will be given in more detail. He was small, well nourished, somewhat pale. The eyes were prominent, both lobes of the thyroid were considerably enlarged, there was a definite von Graefe, no tremor. Pulse 90. Teeth were in poor condition, considerable pyorrhea

and several crowns. The tonsils were injected and buried. The lungs showed a few coarse moist râles scattered throughout both sides. The heart was normal in size, sounds were clear. Blood-pressure was 130. The abdomen was negative. The hands at the time of examination were edematous; this edema appeared the night before. There was no edema of the ankles. All deep reflexes were active. The urine was negative. Blood examination: red cells 5,432,000, leukocytes 26,250, hemoglobin 85 per cent. Differential: polymorphonuclears 30, large lymphocytes 33, small lymphocytes 29, eosinophils 8. Coagulation time 5.5 minutes. Skin tests for protein sensitization to some of the commoner proteins gave a slightly positive wheal for chicken and wheat. *x*-Rays of the teeth showed several pus pockets (apical abscesses). Stool examination was negative.

The high white count in this case is very striking, and the eosinophilia is the only one in our series in which the total eosinophil count was increased to any degree. In fact, this case stands out on account of the eosinophilia and the high leukocyte count. What the increase in mononuclears signified was not clear. No further studies were made on this patient, and as it seemed at least possible that the condition of the teeth might have some relationship to the edema, it was decided immediately to have dental work done. Immediately after the teeth were drawn the edema which had been present disappeared, and no attacks occurred for two months until a cold in the head was followed by sudden edema of the right foot and leg. On July 28, 1919 the patient was seen again, and reported that the attacks were not as frequent or as severe. On August 24th he again caught cold, and two days later he again had a typical attack of angioneurotic edema. At the time of writing, March 15, 1920, the patient reports a very definite and continued improvement, with very few attacks, and those of minor consideration. Blood examination on March 15, 1920, white cells 11,100; differential: polymorphonuclears 73, large lymphocytes 4, small lymphocytes 20, eosinophils 2, basophils 1.

This case may be an angioneurotic edema with an infectious etiology.

Case IV.—R. E. W. Male, white, married, thirty-five years, seen December 16, 1919. This patient dates all his trouble from December, 1916, when his first attack followed the eating of strawberries. He gives a typical story of angioneurotic edema and says he has been to a skin specialist, has had nine teeth extracted, has discussed the advisability of tonsillectomy, but the only time he noted any improvement was during a vacation in a southern climate for five weeks. His bowels are regular, his general health good, and his attacks, which occur at night usually, are present both in summer and winter. His family and past histories are both negative as far as the patient knows. Examination shows a completely normal young man. Blood examination: Leukocytes 11,900 two hours after his dinner, hemoglobin 90 per cent. Differential: Polymorphonuclears 73, small lymphocytes 17, large lymphocytes 8, basophils 2. In discussing the case with the patient there seemed no possible etiologic agent ascertainable until the patient suggested that he ate an unusually large amount of salt. Otherwise he had been on a diet practically free from animal protein. The possibility of a salt metabolism disturbance being suggested by the patient himself, we tested his renal function with the Mosenthal test diet. The test followed the normal curve in every way except that the excretion of sodium chlorid amounted to 16 grams on an intake of 13 grams. Naturally, this did not prove an actual disturbance of sodium chlorid metabolism, but it at least suggested it, and as further metabolic studies were impossible at the time, the patient was asked to discontinue all dietary precautions and simply to cut down his salt intake to approximately 10 grams a day. *Post hoc* if not *propter hoc*, the patient remained free of attacks of edema for at least three months.

Case V.—Mrs. H. S. Female, white, married, forty-three years old. Urticaria. Seen in September, 1919, during an attack of hives which had lasted three weeks, and which had followed the eating of scallops and Camembert cheese. Her hives were worse at night and big welts appeared when she took off her clothes and lay in bed.

Past History.—For the past five years has had attacks of hives. Several months ago she had some ovarian disturbance, for which Dr. Baer gave corpus luteum. After every tablet menstruation immediately followed, because of which she stopped taking the tablets. She is an easily excited woman, but has noticed that she never sweats, and that things and conditions which seemed to make other people perspire merely irritated her skin. Otherwise she has been a perfectly normal, healthy, active woman, quite busily engaged in the many activities of the modern lady. No data on the family history of any special value were obtained. Examination showed a stout, rather dumpy woman. The hair and skin were very dry, the throat and teeth were in perfect condition, the thyroid was not palpable. Heart and lungs were normal. Pulse 72. The skin all over was almost covered with giant hives which showed the scratch-marks of severe itching. Protein skin tests for some of the commoner substances were negative.

On seeing this patient, what struck one immediately was the dryness of the skin and hair, the absence of sweating, the rather unusual reaction to corpus luteum, the adiposity—and the hives. We guessed that there might be some endocrine disturbance present, so we decided to try thyroid extract. The patient was put in bed with many hot-water bottles inside the bed, but with the bedclothes lifted off the skin by a frame. Within twenty-four hours 25 grains of thyroid extract were administered under careful supervision, and within forty-eight hours the patient had begun to show a disappearance of the hives. In seventy-two hours they had all cleared up. This may not have been due, of course, to the thyroid extract, as the time for the cessation of the attack may well have arrived. However, subsequent history, when she was continually under small doses of thyroid extract, at least indicates, if not proves, that before the administration of thyroid extract she was suffering from a mild hypothyreosis, for during the two months following the terrific attack of hives, the patient under thyroid extract volunteered the information that she was feeling like a new woman in every way and had more "pep" and energy than she had had for a long time.

DISCUSSION

Needless to say the cases chosen to be presented to you are only those in which some etiologic agent seemed present. The histories are all briefly sketched. Despite every effort, most of my cases revealed no open lock into which to fit a therapeutic key, and are therefore not presented. Perhaps the cases presented, however, may give us a clue for the discussion of all of them.

In the first place it is a rather striking paradox in my series that almost all patients date their attacks of chronic hives or angioneurotic edema from some food poisoning. Yet in not a single case was I able to demonstrate either by the skin sensitization tests or by clinical investigation a real causal connection between food and the attacks. Unlike the acute hives, which so frequently seem to be an indication of an anaphylactic state toward some specific food protein, chronic hives in all but two of my cases seem to be uninfluenced by any dietary regulations. Case I was definitely associated with a protein intoxication, but even here there was some other underlying factor. In Case V the investigation was not intensive enough to admit definite statements, yet it does seem probable that the high salt intake may have had a great deal to do with the condition. It was rather striking that eosinophilia, which is such a common accompaniment of the anaphylactic state, was only found to any extent in one patient of our series, and this patient seemed to be improved when a focal tooth infection was removed. I must confess to being surprised at the absence of eosinophilia, and yet this fact does harmonize with the contention that food plays a minor rôle in the etiology of either chronic urticaria or angioneurotic edema.

A word should be said about other possible metabolic disturbances. As is well known, calcium often has a beneficial effect in these cases, and in one of our patients a low calcium content was found in both blood and urine. Other possible metabolic disturbances might occur. So little is known of inorganic chemistry of the body, and the technical difficulties of studying inorganic metabolism outside of a hospital are so

great, that it has been impossible in this series to go into this matter. As far as I know no conclusive work has been done on the organic blood chemistry of these cases. The studies of Miller and Pepper show a nitrogen retention during the attacks greater than in the free period; second, that the total acid excretion and the ammonia excretion are normal; third, sodium chlorid excretion was reduced preceding the attack and increased after an attack; fourth, the low chlorid intake had a beneficial effect on their case of angioneurotic edema. However, their patient showed some evidence of nephritis, and for that reason their conclusions cannot generally be made to accommodate other cases of angioneurotic edema unless further substantiated by studies on pure cases not associated with nephritis.

Focal infections must be discussed, although no general conclusions can be drawn from my series. Some of the patients naturally enough showed definite focal infections, as any series of 25 individuals would, and in at least 2 the condition cleared up when the focal infection was removed. It would seem to me that without being able to prove even in an individual case the causal connection between infection and either urticaria or angioneurotic edema, any such patient possessing a focal infection should be relieved of the infection. The experience cited in Case I as well as Case III brings up the thought that even in conditions of apparent pure protein intoxications there may be an underlying infection present. For instance, diabetes, which is a metabolic disturbance, is made worse by the onset of any acute infectious disease. A physician with recurrent attacks of iritis found that the attacks were always made worse by the ingestion of excessive quantities of meat. Subsequently a general infection of his sinuses was demonstrated.

The last case shown would seem to bring up the discussion of the relation of hives or angioneurotic edema to the endocrine system. Of course, the case does not prove the causal connection, and in at least 3 others of my series there were present signs of hyperactivity of the thyroid gland. It would seem wise to investigate patients with chronic hives for disturbances of the endocrine system, and the new Benedict portable respira-

tion calorimeter may help in clearing up the etiology of some patients.

The neurotic temperament seems to be present in almost all cases. If, as we think, chronic urticaria and angioneurotic edema are different manifestations of the same fundamental disturbance, then surely the neurosis must play a big element in both. Just as in acute hives the nerve control of the smaller blood-vessels of different individuals varies in such a way as to make one of many respond to the ingestion of fish by an attack of hives, so, in the chronic condition, it would seem highly probable that an angioneurosis must be present. The history of large families with angioneurotic edema, which have been reported by Osler and Crowder, certainly lends support to this view. What this angioneurosis is in terms of our present conceptions is not known. Our second patient seems to be an illustration of an almost pure neurosis. No therapy can hope to be successful unless the general hygiene of the patient is improved and his general strength built up.

The studies of Osler remain classical. He groups both these conditions under the general heading of the erythema group of skin diseases, because on clinical grounds they seem to be so closely related. The systemic manifestations of the erythema group of diseases were emphasized by Osler, and may vary from an edema of the larynx or gastro-intestinal mucosa to a hematuria or hemarthrosis. In the series of patients under discussion no systemic complications are noted, but in an earlier series studied in conjunction with Dr. Louis P. Hamburger, of Baltimore, Maryland, the gastro-intestinal disturbances were particularly investigated. Although the notes on this study are no longer available, certain conclusions are remembered.

The abdominal crisis of the erythema group may simulate an acute appendicitis and intussusception, or may not be typical of any definite pathologic condition. In our cases the attacks occurred usually at night, more often in the early morning hours; and in the beginning at least kept one guessing as to the exact condition. Localized symptoms and signs might occur, fever and leukocytosis might be present, and yet usually there was

something which did not quite fit in the clinical picture of the suspected lesion. It happened in several cases that during the period of observation an attack of urticaria or erythema occurred and cleared the diagnosis; or that the patient or his family volunteered the information that the patient was susceptible to a skin disease. This information was often helpful in averting operation, and in one case at least only the keenness of Osler himself prevented an unnecessary operation for intussusception. Several patients had been operated on without a diagnosis having been made; in fact, one patient had had at least two exploratory laparotomies before the diagnosis of angioneurotic edema was established. Subsequently this patient developed an attack of acute suppurative appendicitis.

Although the visceral manifestations of this group of skin diseases seem to be comparatively rare, it is well to bear in mind that at any time a patient with either recurrent urticaria or angioneurotic edema might develop a fatal edema of the larynx or an abdominal crisis, non-surgical in nature, which in many ways simulates a true surgical condition. Naturally, surgical intervention would be contraindicated. On the other hand, the review of the Baltimore cases showed that occasionally even a patient with angioneurotic edema might develop a true attack of appendicitis.

In conclusion I should like to say a few words regarding my impression of the general nature of these cases. That hives and angioneurotic edema are expressions of an identical etiologic agent in every instance seems highly improbable from the evidence so far collected. Both urticaria and angioneurotic edema are to be considered symptomatic expressions. Often the fundamental etiology is clouded in mystery. At times an associated condition, such as disturbance of the endocrine system, a marked neurotic predisposition, an infection, or a metabolic error, suggests possible etiology. Therapy directed toward the correction of the suspected cause, even if successful, does not prove that the sequence is one of cause and effect, since both diseases are in their very nature periodic in character.

These patients suffer intensely, and after they have had their

illness any length of time are willing to go to almost any extreme in the effort to effect a cure. How, then, are they to be handled? If the conception of the illness just expressed is correct, the natural sequence would be as follows: Each patient with chronic urticaria or angioneurotic edema is an individual problem. It is not practical to put every such patient "through the paces," but often a carefully taken history and physical examination will suggest a line of investigation to be followed. Focal infection should be eliminated for reasons given in the body of this talk. Glaring dietary indiscretions should be corrected, and further investigation suggested by the history or the physical examination should then be made. Such patients usually do not enter the hospital. If they did, thorough metabolic studies might clear up some of the mystery. Still, if a metabolic error is suggested, the patient should have the benefit of the doubt and a chemical equation should be worked out as far as possible. I believe that if this plan were generally followed more of these patients would become free from their symptoms.

Note to Case I.—This patient was under the joint care of Dr. Solomon Solis-Cohen, of Philadelphia, and myself, and it is through his courtesy that I am permitted to include this case in my series.

CLINIC OF DR. JAMES G. CARR

Cook County Hospital

BRONCHIECTASIS WITH PULMONARY HEMORRHAGE

We want to discuss with you today the case of a patient who was in the hospital last summer, who was sick with a pulmonary infection, died after several severe hemorrhages, and went to autopsy.

The patient was a Polish laborer, thirty-two years of age, unmarried, who was admitted to the hospital July 15, 1919. His chief complaint was of pain in the left side; with this he had a cough, was weak, and had lost weight. Two weeks prior to admission he was taken with a sharp pain in the left side, which was constantly present, aggravated by coughing and sudden change of position. It had no relation to the taking of food. The cough was present from the onset of the pain and was more marked in the morning than during the day or night. There was a tough, greenish sputum with a very foul odor. There had been no expectoration of blood. The weakness was progressive from the onset of the sickness, and the patient stated that he had lost some 15 pounds in weight. The appetite was poor; the bowels regular.

In September, 1918 the patient had influenza; he was sick for six weeks, about two of which were spent in a hospital. Since that time he has not been entirely well, but worked up to the date mentioned as marking the onset of his present illness. It was difficult to get an accurate history from the patient, but he obviously felt that his illness was the result of the influenza referred to. There was no history of any other disease. Venereal disease was denied. Alcohol had been used in moderation, tobacco to excess. The family history was negative.

On admission the temperature was 103.6° F., pulse 84, respiration 30, blood-pressure 135 systolic, 80 diastolic.

Physical examination showed a well-developed, rather robust man. A troublesome cough was noted, with foul-smelling sputum. The right pupil was small and irregular; both pupils reacted to light and accommodation. Pyorrhea was marked. A friction-rub was present over the base of the left lower lobe in the anterior and midaxillary regions. Over a limited area in the left axilla, from the fourth to the seventh ribs, there was a hyperresonant to tympanitic note. Vocal fremitus was exaggerated in both axillæ. Below the spine of the left scapula, toward the midline, there was a "dollar-sized" area over which numerous subcrepitant râles could be heard. Over the left base posteriorly there was dulness; the breath sounds were distant, fremitus was unimpaired, and a few râles were heard. The heart was normal except that the tones were distant and somewhat indistinct. Abdominal examination was negative. The extremities were negative. The reflexes were normal except for some sluggishness of the patellars.

Laboratory Findings.—The urine showed a specific gravity of 1026, was alkaline in reaction, contained neither albumin nor sugar. There was a leukocyte count of 25,700. The report from the roentgenologist on July 19th runs thus: "Screen findings are negative for indications of pulmonary tuberculosis." The blood Wassermann reaction was negative.

On July 21st it was noted that "the temperature has been normal for two days; the cough is much less and the patient is up, feeling much better." On July 24th "cough and offensive sputum, with temperature, continue." On July 26th the leukocyte count was 12,800 and the history sheet reads, "Patient expectorates about 300 c.c. of foul sputum in twenty-four hours." Sputum examinations were made frequently, but tubercle bacilli never were found. On the 18th and 20th of July elastic fibers were noted. The report for July 20th reads, "The sputum has an offensive odor; is formed in three layers; contains elastic fibers, leukocytes, mainly polymorphonuclear in type, though great numbers of small mononuclears are present. No tubercle

bacilli are found." A second *x*-ray examination was made July 29th, and the report reads, "There is no *x*-ray indication of a pulmonary abscess or similar lung change. Pulmonary areas are all clear. It is noted that in the lower left thorax there is a slight loss of clear detail, which is probably evidence of pleural pathology of low-grade extent."

In making a diagnosis we sought to determine the nature of the anatomic lesion present and the etiologic factor producing the lesion. Anatomically our physical findings warranted a diagnosis of bronchopneumonic infiltration of the left lower lobe. We will discuss later the question as to the possible existence of a lung abscess or bronchiectasis within this pneumonic area. Etiologically, we first tried to reach a conclusion as to the tuberculous or non-tuberculous nature of the process. Against the diagnosis of tuberculosis we had: (1) The location of the lesion. Tuberculosis is rarely primary in the lower lobe, and neither physical examination nor the *x*-ray had produced evidence of a primary lesion elsewhere. (2) The leukocytosis. (3) The constant failure to find tubercle bacilli in the sputum. We could not regard this bronchopneumonia as tuberculous in type, and though the warning may be trite, we would emphasize the importance of searching for some other cause than tuberculosis in any case of supposed tuberculosis with abundant sputum and a constant absence of tubercle bacilli.

Syphilis of the lung is more common than has been generally supposed and deserves more frequent consideration. The negative history and serum reaction, the comparatively minor physical findings in relation to the patient's general condition (in pulmonary syphilis the general condition is often very good, even in the presence of extensive pulmonary findings), the absence of any stigmata of syphilis, save possibly the inequality of the pupils, made the diagnosis of syphilis unwarranted.

If, as it appeared, we were dealing with a non-specific infection, what was the nature of the lesion? Was it a pulmonary abscess, a bronchiectasis, or an unresolved pneumonia? And, finally, did the lesion result from the influenza which had occurred ten months before?

The question of unresolved pneumonia would better be disposed of first. Either the primary pneumonia must have occurred in September, 1918, or within two weeks of the patient's recent admission to the hospital. In regard to the former possibility, an unresolved pneumonia of ten months' duration, without acute symptoms during that period, would probably mean a chronic fibrosis, under which hypothesis we could not explain the acute symptoms that existed when the patient came under our care; these could only be caused by an active process to which the term "unresolved pneumonia" was not applicable. As to the possibility of an unresolved pneumonia following an acute process shortly before we saw the patient, the history he gave us was not that of pneumonia, as the term is generally used. The high temperature and the leukocytosis two weeks after the onset of the disease point to some present acute process, not properly included under the term "unresolved pneumonia." and the quantity and character of the sputum were further obstacles to the acceptance of such a diagnosis. "Unresolved pneumonia" in general is a diagnosis which ought only to be accepted when other possible diagnoses can be definitely excluded. In this case the character and quantity of the sputum and the constitutional symptoms of an acute infection made it imperative to search for a more satisfactory diagnosis.

We came to the differentiation of pulmonary abscess and bronchiectasis; the one, under certain conditions accessible to surgical treatment; the other, calling for medical or climatic treatment. In favor of the diagnosis of abscess of the lung we have the fever, the constitutional symptoms, the leukocytosis, and the character of the sputum, especially the presence of elastic fibers. Bronchiectasis is usually a chronic process. Acute symptoms must be explained if the case is diagnosed as bronchiectasis. This also applies to the presence of such a leukocytosis as occurred in this case. Drainage is usually fairly good in bronchiectasis, and leukocytosis as a result of septic absorption is not likely to occur. Elastic fibers may occur with bronchiectasis when ulceration of the bronchial walls has taken place.

The physical findings and the results of *x*-ray examination

were not those of abscess. The signs of cavity were not present; the hyperresonant to tympanitic note described in the left axilla at the first examination was never very distinct; it was not suggestive of a cavity and was probably the result of an inflammatory edema about the area of bronchopneumonia. There was no change in the percussion note after the discharge of large quantities of sputum. But the absence of a tympanitic note over the area most involved, of amphoric breathing, of a change from dulness to tympany after a paroxysm of coughing, with the expectoration of much sputum, are all compatible with the existence of a small abscess deep in the infiltrated lung. We were thrown back upon the history. More and more we became convinced that the existing disease had not developed in the two weeks immediately preceding our acquaintance with the patient. We believed that whatever condition was present had been related to the attack of influenza mentioned. Whether abscess or bronchiectasis, it was the result of pulmonary infection of ten months' standing. Now an abscess of such duration might be expected to show the physical findings and the *x*-ray features typical of that condition. Moreover, an active abscess should have incapacitated this patient much earlier than had occurred; the symptoms of sepsis would have been more pronounced. The most plausible explanation of the condition appeared to be this: there was a bronchial dilatation ensuing upon the influenza, from which point as a focus a bronchopneumonic infiltration had started, resulting in an extension of the infection to the pleura about two weeks before the patient's admission to the hospital. We accepted the diagnosis of bronchiectasis with subsequent bronchopneumonia, and finally, fibrinous pleurisy.

On July 31st, about 10 P. M., the patient had a pulmonary hemorrhage; about 50 to 75 c.c. of blood were coughed up. Hemorrhages of small amounts occurred on August 1st and 2d. On August 4th there was a violent chill lasting about ten minutes. Following this chill the rectal temperature was 107° F. For several days prior to this the temperature had ranged from normal to 101.6° F. The hemorrhages continuing, Dr. H. W.

Gray was asked to collapse the lung. This was done on August 6th, 700 c.c. of nitrogen being injected. On the following day 12 c.c. of whole blood were injected subcutaneously. In spite of these measures severe hemorrhages continued. August 9th another intrapleural injection was made, this time 750 c.c. of nitrogen being injected. The patient did not improve and death occurred on August 10th.

Autopsy Findings.—The autopsy report included the following: (1) Over the left lower lobe the pleura was very thick; there was no effusion.

(2) The upper lobe of the left lung was collapsed.

(3) About one-half of the lower lobe was firm, entirely airless, and about the size of the fist. The weight of the lower lobe was 920 grams. On cut section there was seen an area of consolidation covered with mucopus. The bronchi also contained pus. There were extensive scattered and confluent hemorrhages in the parenchyma.

(4) Within the area described there were several bronchiectatic cavities, the largest the size of a small walnut. This largest cavity was occupied by a recent blood-clot. Cultures from this cavity showed no growth.

(5) A fresh pneumonic area was present about the old one.

The features of especial interest in this case are: 1, The probable source of this patient's trouble at the time of his admission to the hospital was the bronchiectatic cavity discovered at autopsy. About this the lung tissue had probably never become normal after his influenza. A latent process had become active and manifested itself in the onset of a pleurisy about two weeks before his admission to the hospital. Evidence of pneumonic change of long duration, "unresolved pneumonia," was present, but as a consequence of infection originating in the dilated bronchi. Whether the pneumonic process had never entirely resolved after the attack of influenzal pneumonia, or, having resolved, had developed a second time about the bronchiectatic focus, we cannot say. We can say that the autopsy disclosed a very obvious reason for the existence of this "unresolved pneumonia."

2. Theoretically, collapse of the lung should have stopped the hemorrhage in this case; lung tissue compressed about the cavity discovered should have made a tampon of the contained clot. Practically, the pleural adhesions and the pneumonic lung prevented the collapse where it might have been of therapeutic benefit. Only the uninvolved portions of the left lung were collapsed. Our therapeutic failure was explained by the conditions found at autopsy.

3. We regret our inability to show you the *x*-ray plate, which cannot be found. You will note, however, that the second plate, taken twelve days before death, showed a "slight loss of clear detail." Perhaps the process was more acute than appeared at autopsy.

CHOLELITHIASIS WITH CHRONIC JAUNDICE

THE patient whom we present to you this morning was admitted to the hospital January 17, 1920. He was born in Switzerland forty-four years ago, is unmarried, and a porter by occupation. Eight years ago he had his first attack of pain in the right upper quadrant of the abdomen. The pain was very severe; for two years he had attacks at intervals of a few weeks. These attacks lasted three or four days; jaundice was present a large part of these two years; it was aggravated at the time of the attacks of pain. The patient is sure he has passed many gall-stones. During the ensuing three years he had little trouble, but for the last three years he has had attacks at intervals of two to eight weeks.

The attack begins with the sudden onset of pain, sharp, sticking, and severe, which usually lasts twelve to eighteen hours. This pain radiates to the right side, but not as far as to the shoulder, nor does it radiate to the thigh or genitalia. Jaundice appears a day or two after the pain and is most intense twenty-four to forty-eight hours after its first appearance; it then slowly recedes. For some time these attacks have been so frequent that the jaundice is never entirely absent. The stools are grayish white during the attack; the urine looks "almost like ink"; there is always a rather dark color to the urine. Vomiting occurs with every attack of pain; the vomitus is green and bitter. The patient has lost about 50 pounds in the last eight years. There is marked weakness at the time of the attack; if the interval between the attacks is sufficiently long the patient becomes able to work, but he is never really vigorous.

There is an indefinite history of "rheumatism" years ago; otherwise the history is negative. Venereal disease is denied. The patient has used alcohol and tobacco in moderation. The family history is negative except that the mother, who died of consumption, was subject during many years to attacks of gall-stone colic.

Physical examination shows a small, poorly nourished man of middle age, who is deeply jaundiced. The eyes react to light and accommodation; the scleræ are yellow. Cardiac examination is negative. Over the right apex posteriorly there is some impairment of resonance, with increase of fremitus and of the whispered voice. Liver and spleen are both palpable; there is considerable resistance to palpation over the hepatic area, but the edge and surface of the liver appear smooth. The edge of the liver is about three fingerbreadths below the costal margin in the mammary line. There is a slight swelling in the region of the gall-bladder. There is no evidence of ascites. The temperature has been normal most of the time, but at least once a day has gone as high as 99.5° F., and some readings of 100.4° F. are recorded. The pulse-rate is about 80, respirations about 20.

Laboratory Findings.—The urine shows a specific gravity of 1018 and contains albumin and bile, with hyaline and granular casts. Blood examination shows a hemoglobin percentage of 45, 3,370,000 erythrocytes, and 3300 leukocytes. The differential white count was not significant; the red corpuscles are stated to have been "fairly normal." Gastric analyses on successive days, by means of the Ewald breakfast, showed a free HCl of 9, a total acidity of 40 in one instance, and in the other a free HCl of 13, and total acidity of 54. Mucus was present in considerable quantity, but no blood was found. Wassermann reaction was negative on blood and spinal fluid.

***x*-Ray.**—The reports from the roentgenologist read: "There are no *x*-ray indications of definite pulmonary pathology. Isolated areas of calcification indicate obsolete areas of tuberculosis. No marked degree of fibrosis is indicated. The median shadow reveals a fusiform bulging, probably an incipient aneurysm of the lower portion of the descending thoracic aorta; and no evidence of a gastric or duodenal lesion is noted. The stomach occupies a median position, as though displaced to the right, possibly by an enlarged spleen. The duodenal bulb seems pushed to the left, as though by an enlarged liver or gall-bladder. No gall-stone shadows noted."

The essential problem presented in this case is the diagnosis

of the cause of the icterus. The answer to this problem will, in all likelihood, be found to be the answer to all the questions brought up by the history and physical examination. The history points to a so-called "obstructive jaundice," by which we mean an interference with the passage of bile through the larger ducts. But in the stricter sense we now regard all jaundice as obstructive. The obstruction may be—

(1) Of the larger ducts, the result of foreign bodies or products of inflammation, such as mucus or swollen mucous membrane, within the ducts; of new growths or inflammatory conditions, with contraction in the wall of the ducts; or of tumor, enlarged glands, or diseased organs producing pressure thereon.

(2) Of the finer biliary passages within the liver, through the deposit of syphilitic granuloma or the scars thereof, or multiple malignant metastases actually blocking the smaller ducts; or through the production of an abnormally viscid bile, which may occur through acute and chronic destruction of the red corpuscles, or in degenerative conditions of the liver; or through the obstruction of the finer biliary passages and their practical obliteration by the débris of degenerated hepatic tissue, or the deposit of chronic inflammatory tissue with or without contraction.

The problem of diagnosis here is to determine whether the obstruction is of hepatic origin, or whether it arises in the larger ducts. When we make use of the term "obstructive jaundice" in the discussion, as evidenced, for instance, by "clay-colored" stools, we refer to the form of obstruction which prevents the flow of bile through the larger biliary passages; practically this means the common duct. Stone in the cystic duct, theoretically, ought not to cause jaundice; if such occurs it is because of associated inflammation, or possibly through the pressure of a large stone upon the common duct. Obstruction of the hepatic duct by stone is rare. The stone is likely to find its way to the narrowed terminal portion of the common duct.

A correct diagnosis is of prime importance in the presence of icterus; where possible it is essential to relieve the icterus by removing the cause. It is of equal importance not to undertake

a needless surgical procedure in an icteric patient. Once you have seen the persistent oozing of blood which sometimes sets in after such an operation, and which not infrequently proves uncontrollable, you will thoroughly appreciate any words of caution about these cases, and the importance of taking all possible care to be sure you are right before you go ahead. In this instance the temptation is strong to accept the obvious diagnosis of stone with suppuration in the common duct and advise operation. The point of the observation just made is to avoid this haste. Let us first consider the possibilities of this case and build our diagnosis with due consideration of these possibilities.

As the case now stands, we have jaundice of long standing, associated with a history of pain coming on in attacks, an enlarged, probably smooth, liver, a tumor in the gall-bladder region, an enlarged spleen, a secondary anemia of moderate severity, a steady loss of weight, bile in the urine and stools, diminished gastric secretion, and negative Wassermann reactions on the blood and spinal fluid. We have, further, a history in the earlier course of the disease of the onset of jaundice after the attacks of pain; in the later period of the illness the jaundice has been constant, but has deepened after the paroxysms. The history also includes the statement that "clay-colored" stools occur after the attacks. These statements must be confirmed by observation.

According to Rolleston, syphilis of the liver may simulate: (1) Portal cirrhosis; (2) lardaceous disease; (3) tumor of the liver; (4) hepatic suppuration; (5) cholelithiasis; (6) splenic anemia; (7) hypertrophic biliary cirrhosis. We might add that secondary syphilis is occasionally associated with enlargement of the liver and spleen with jaundice. It is idle to take up in detail the differential diagnosis of the case before us from each of the types mentioned. This patient manifestly does not present a jaundice of some eight years' standing which is caused by secondary syphilis. Neither have we a symptomatology like that of lardaceous disease, nor of splenic anemia, nor of the later stage of splenic anemia known as Banti's disease. Hepatic

suppuration may be simulated in a striking manner by syphilis of the liver, but this case presents no similarities to sepsis. We will discuss shortly Hanot's cirrhosis, or hypertrophic biliary cirrhosis; the symptom-complex resembling this condition caused by syphilis can only be differentiated from the true Hanot's disease by the characteristic diagnostic features of syphilitic disease in general, which are: the history, the serum test, the presence of evidence of an earlier syphilis—such as scars or general adenopathy and the response to antisyphilitic treatment. We can group together the three types under one of which this case might be classified if demonstrated as syphilitic, and state that the absence of any positive evidence of syphilis is the one feature of the case which makes such a diagnosis untenable now. Furthermore, the absence of ascites in a case of syphilitic hepatitis or cirrhosis of such long duration as has happened here is not to be expected; we have no physical findings of tumor of the liver or of scars of old gummata. We must depend on general diagnostic features to differentiate a true cholelithiasis from a similar symptom-complex the result of syphilis, though the frequency of the one as compared to the rarity of the other is of considerable importance. As a general observation regarding the diagnosis of hepatic syphilis, we may say that too much reliance is not to be placed on the history and serum reaction; no other late results of syphilis are more likely to be negative in these particulars than those involving the liver. In fact, the diagnostic procedure formerly so widely employed of noting the response to treatment may be said, without exaggeration, to be of prime importance in making a certain diagnosis in hepatic disease. The question of the specific or non-specific nature of a given hepatic disease frequently turns finally on the response to antisyphilitic treatment. This patient has already been given potassium iodid; he will be kept on this medication with the addition later of mercury in some form, if he has no recurrence of the paroxysms of pain. The mercury will not be added until he has been kept on iodid for a few weeks and another Wassermann test has been made. Since thus far no positive evidence of syphilis can be produced, the occurrence of a paroxysm of

pain, such as the patient has described, with deepening of the jaundice and clay-colored stools, will not justify us in keeping the patient longer on specific treatment. We cannot accept a diagnosis of syphilis on present evidence, though we cannot deny its possibility. We will employ specific therapy as a part of the observation of the case. However, a confirmation of the patient's statements regarding the paroxysm and the sequence of the symptoms will be convincing evidence to us of the non-specific character of his disease.

Jaundice with an enlarged liver and a history of loss of weight in a patient of this age suggests malignant disease. We have to consider three types of cancer as possible causes of this jaundice: (1) Carcinoma, causing obstruction of the common duct; the new growth may be in the head of the pancreas, the duodenum, or the bile-duct itself. Each of these conditions produces a deep jaundice which, once established, does not show partial remissions. The stools are devoid of bile and the emaciation is much more rapid than has been the case in the patient before us. These conditions can be excluded as causing a jaundice of this duration. We will need to take up for further discussion the question of cancer of the bile-duct, following long-standing irritation from stone.

(2) Primary malignant disease of the liver is rare, and it is hardly conceivable that we could thereby explain the symptomatology of this case. Cancer of the liver of any type will not produce a jaundice of three years' standing, to say nothing of the previous series of attacks dating back eight years and lasting two years or more. Jaundice due to malignant disease of the liver never lasts three years. The smooth surface of the liver and the absence of ascites are further objections to the diagnosis of cancer of any type.

(3) Secondary malignant disease is the common form of hepatic cancer; the most common primary sites in men are the stomach, rectum, and large bowel. The *x*-ray is negative for any evidence of gastric carcinoma, and gastric analysis shows free hydrochloric acid without retention, blood, or other evidence of cancer. Rectal examination is negative. No other reasons

have been found supporting a diagnosis of cancer of the rectum or large bowel. No other primary disease has been discovered. Moreover, if the onset of the jaundice three years ago was the result of malignant disease, our patient should long ago have departed this life. When jaundice occurs as the result of secondary malignant disease the condition may be regarded as far advanced.

Though we can dismiss cancer as the cause of this jaundice, we cannot assert the entire absence of malignant disease. Long-standing irritation, the result of stone and inflammation, may have been succeeded by a carcinomatous process in the bile-ducts; such a process may be a factor in the production of the jaundice now, but it is not the only factor, and it has not been the cause of the jaundice of which this patient complains. We can neither affirm nor deny the presence of carcinoma of the bile-ducts or gall-bladder; if present it is not the original cause of the symptoms, and operation alone will settle the question of diagnosis.

There are three conditions left for consideration:

(1) Hanot's cirrhosis, a disease characterized by jaundice of long duration, enlargement of the liver and spleen, paroxysmal attacks of pain in the hepatic region, the presence of bile in the stools, and the absence of ascites. The condition is oftentimes spoken of as hypertrophic biliary cirrhosis, a term which is not strictly synonymous with Hanot's disease. Hypertrophic biliary cirrhosis occurs as a result of chronic phosphorus-poisoning, and as a late result of chronic infectious processes producing cholangitis. The clinical differentiation from Hanot's disease rests substantially on three points: first, the presence of a definite cause of biliary cirrhosis, such as chronic infection; second, the enlargement tends to be followed by atrophy, while the liver of Hanot's cirrhosis progressively enlarges; third, the occasional absence of bile from the stools, which does not occur in Hanot's cirrhosis. The great rarity of a true Hanot's cirrhosis should make us very careful in accepting such a diagnosis. Not long since I heard an experienced pathologist remark that Hanot's cirrhosis is occasionally diagnosed at the bedside, but rarely

at autopsy. It is true that in this case we have the cardinal findings in Hanot's cirrhosis except for the history (not yet proved) of clay-colored stools following the attacks of pain. This is an essential point; if our observation confirms this history we are warranted in refusing to diagnose Hanot's cirrhosis.

(2) Hemolytic family icterus of the acquired or Chauffard type. The long-standing jaundice with the enlarged liver and spleen and the anemia suggest this condition. As the name implies, this disease is hemolytic in origin; an increased fragility of the red blood corpuscles is a characteristic of this clinical entity. The fragility test has been made, with a negative result. There is slight hemolysis with a sodium chlorid solution of 45 per cent., which is normal.

(3) We are back, finally, to the question of obstruction of the common duct by stone. This may have resulted in one of three ways: (*a*) The intermittent passage of stones from the gall-bladder, assuming that the latter contains many stones. Thus we might account for the attacks of colic, occurring with such frequency that the jaundice does not disappear in the intervals. (*b*) Impaction of the stone in the common duct. There is no justification in the symptomatology for the diagnosis of the intermittent hepatic fever of Charcot which is characterized, according to Osler, by "ague-like paroxysms, chills, fever, and sweating," with long-standing jaundice of varying intensity and pain with the paroxysm. The distinguishing signs of stone in the common duct, as laid down by Naunyn and quoted by Osler, are: "(1) The continuous or occasional presence of bile in the feces; (2) distinct variations in the intensity of the jaundice; (3) normal size or only slight enlargement of the liver; (4) absence of distention of the gall-bladder; (5) enlargement of the spleen; (6) absence of ascites; (7) presence of febrile disturbance, and (8) duration of the jaundice for more than one year." The case before you answers to all these conditions except the third and fourth. We will shortly discuss more at length the enlargement of the liver and the tumor noted in the region of the gall-bladder.

(*c*) A stone once impacted in the common duct may have

formed a diverticulum in which it usually lies, passing, from time to time, into the lumen of the duct and causing obstruction. Such a case we saw once last spring at Mercy Hospital. The patient was admitted to the hospital with jaundice and the history of recurring paroxysms of pain. Ten weeks prior to admission she had been operated in another city for gall-stones, and she came to us with a report that no gall-stones were present, as none had been found at operation, and a fairly large sound had been passed through the cystic and common ducts. Six weeks subsequent to operation the attacks of colic for which the operation had been undertaken began again and jaundice supervened. She was operated at Mercy Hospital by Dr. Neff and a stone of considerable size was removed from a diverticulum near the lower end of the common duct.

Before we accept the diagnosis of stone we must dispose of certain objections to such a conclusion which may be drawn from the results of our examination:

1. The enlargement of the liver needs explanation, especially in view of the statement quoted from Naunyn. We need only restate what was said in the discussion of Hanot's cirrhosis concerning hepatic enlargement as the consequence of long-standing infection of the bile-ducts. Such enlargement of the liver does occur in the course of a prolonged cholangitis.

2. The low leukocyte count. In many chronic infections there is an absence of a leukocytosis. When an infection has been active for some time it may be that the organism loses the power of response to the stimulus which is effective in producing the leukocytosis. In infective conditions, for instance, in chronic endocarditis of a low-grade or subacute type leukocytosis is not always present. Again, in the present case, we do not know that there is no leukocytosis at the time of the paroxysm and immediately thereafter. At present we may assume as a possible explanation that drainage is good, and no retention being present, there is no leukocytosis.

3. The enlargement of the gall-bladder (if such is the correct interpretation of the tumor noted in the gall-bladder area and suggested by the *x*-ray), according to the oft-quoted Courvoisier's

law, is evidence of carcinoma, especially of carcinoma of the head of the pancreas. However, it has been abundantly proved that chronic inflammatory conditions cannot be excluded because an enlarged gall-bladder is present.

4. The anemia may be the result of the prolonged jaundice, of chronic infection, or of the nutritional disturbance incident to the prolonged disease of the liver.

The probability is that we are dealing with a jaundice produced by a stone in the common duct. No other diagnosis is so plausible. As to the location of the stone, to choose between the three possibilities already discussed is difficult, if not impossible. We incline to the belief that there is a stone in the common duct without suppuration; the infection is a low-grade rather than a virulent septic process. As to the question of cancer, only an operation can determine whether or not a primary carcinoma of the bile passages is present as a result of long-standing calculous disease. We do not believe that any extensive carcinomatous process will be found. The patient will be kept under observation. As we have stated, specific treatment will be employed unless the course is interrupted by a return of the paroxysmal colic; in which event, if the statements as to the deepening of the jaundice and the clay-colored stools are substantiated, we will advise operation.

February 20, 1920.

I want to report to you this morning on the case of jaundice that you saw four weeks ago today. On January 25th the patient had an attack of acute biliary colic which lasted for three hours. There was no radiation of the pain to the shoulder or back. On the following day the record states that the "jaundice is deepened a little"; the stools were clay colored; chemical examination was negative for bile on the 26th and 27th. On the 29th an attack of excruciating pain began and lasted for thirty hours. Over the two days $1\frac{3}{4}$ grains of morphin were used. The jaundice became very intense. With the paroxysm of pain there was no chill and the temperature was not notably increased. The white count on January 30th was 8200; more than twice the

count of January 18th. Prior to transfer to the surgical service the coagulation time of the blood was determined and found to be five minutes.

Report of Operation.—Operation was done by Dr George F. Thompson February 1st. A mass of adhesions was found about the gall-bladder; in this mass were a few small foci of suppuration. The gall-bladder was freed with difficulty. A large number of stones were removed from the gall-bladder, but none were found in the ducts. There was no suppuration in the biliary passages. There was no evidence of carcinoma. After a somewhat stormy course the patient recovered and will leave the hospital today.

The attacks of colic were evidently due to the passage of stones from the gall-bladder and not to an impacted stone, as we had believed.

CLINIC OF DR. CLIFFORD G. GRULEE

PRESBYTERIAN HOSPITAL

COLIC IN THE BREAST-FED INFANT

WE have here a little baby three weeks old, which case I saw first on March 9th. The history was that the child had been born of a normal labor with a birth weight of 7 pounds, 9 ounces, but that it had failed to gain, and the weight when I saw it then was 7 pounds, 1 ounce. The infant did not cry much during the day, but cried all night long, so much so that the mother and the trained nurse in charge were exhausted. These attacks of crying, which occurred at frequent intervals, were accompanied by frantic movements of the extremities, a hard, tense abdomen, and marked suffusion of the skin with blood. At all times the child had severe eructation and much flatus. The stools were one a day, greenish, with occasional curds. There was no regurgitation and the child was getting 2 ounces of breast milk every three hours, a total of 16 ounces in twenty-four hours, usually sufficient for a baby of this age and weight.

It was very difficult to say just how much the child had gotten previous to this time, but there was no history of overfeeding. On examination, this child was a fairly nourished but small baby; the anterior fontanel was quite wide open and the sagittal suture was soft. Aside from this the examination was negative.

We have here a rather severe colic of comparatively short duration. As to whether this child was overfed earlier there is some question, but it seems very likely that this was not the case. We will come back to this condition later, and I will only state that under treatment given this child the colic had completely

disappeared within forty-eight hours and did not return again during the following week.

The next case about which I am going to tell you is a baby which I saw first on December 12th. She was then six weeks old. She was born of a full-term normal labor, with birth weight of 6 pounds, 3 ounces. She had been fed at the breast every four hours and her weight when first seen was 7 pounds, 6½ ounces. She had been having colic all night every night for four weeks, with some spitting, a great deal of gas, and the stools were only passed when enemas were given. At this time she was obtaining 3 ounces at each nursing.

On examination the child was found to be a fairly nourished infant, and further examination failed to reveal anything abnormal. This child, too, cleaned up on treatment, but much more slowly, as we will see later.

The third case, Baby T., whose record I have here, was born at the Presbyterian Hospital on the 31st of last December. It was a perfectly normal labor, the baby weighing at birth 7 pounds, 13 ounces. The mother was rather an old primipara and the breast milk was slow in becoming established, nor at any time was there a sufficient quantity. On the second day mixed feeding was advised, and this was continued. This supplementary food consisted of half milk and half water, to every 3 ounces of which was added a teaspoonful of dextrimaltose. Varying quantities of this were given, depending upon the quantity of breast milk which the child obtained, and upon the appetite. I first saw the child on the eleventh day. Previous to this time the baby had never obtained more than 7½ ounces of breast milk, and only once that much. The quantity of artificial food which he obtained had been gradually increasing, so that the day before I saw him he had gotten approximately 12 ounces. The weight had not gone up, but had remained practically stationary, the lowest point on the fifth day being 7 pounds, 2½ ounces, and on the eleventh day, when I saw him, he weighed 7 pounds, 3 ounces. The history was that there had been some regurgitation, but for over a week the child had cried incessantly at night. This cry, on careful observation, was seen to be due to

colic, since it was accompanied by much gas, some regurgitation, and the manifestations mentioned in the former history. The number of stools was not increased.

The child on physical examination was rather thin, but otherwise showed nothing abnormal. This child, too, cleared up in a remarkably short time, but it seems to me that before we take up the treatment in these cases it would be much better to discuss the nature of colic and its causes.

I know of no condition comparatively mild in nature which can cause so much disturbance in a household as colic in the baby, but, on the other hand, I wish to warn you against taking the attendant's word for its presence. Many a child is stamped with the stigma of colic whose chief cause of distress is brought about by undue handling and improper care, combined with its own neurotic tendencies. Especially beware of the grandmother, who almost invariably spoils the grandchild if she gets a chance. Or of the neurotic mother, to whom every molehill is a mountain; or, inquire carefully to find out whether a domineering father, in his supreme egotism, has not asserted that all the child needed was to be picked up, and this, when done frequently at his command, is done thereafter at the urgent request of the baby. But when the baby is spoiled its cry ceases when it is picked up, while in the colicky baby, though it may cease for a few seconds, it returns almost immediately.

After we have determined that the infant is affected with that peculiar condition known as colic, it is well to endeavor to understand what colic really means. Colic is a condition caused by irritation of the mucous membrane of the intestine producing an increased formation of gas and a spasm-like contraction of portions of the intestine. The results of these two factors are segments of bowel temporarily walled off in which large quantities of gas have accumulated. Colic is a condition which is most frequently met with in the breast-fed infant. It is a well-known fact that the breast milk favors the fermentative rather than the putrefactive action in the intestine. It is also a well-recognized fact that the fermentative products under normal conditions are not irritating to the intestinal wall. There are in the breast

milk two substances which favor fermentation—the lactose and the fat. Of these two substances there is very little variation in the quantity of the lactose, but the most variable constituent of the breast milk is the fat. This constituent is especially increased toward the end of the nursing period, and its proportion is very high when the quantity of milk is quite low.

Bahrdt and his co-workers have shown that the substances produced by fermentation, which are most active in an irritative way to the intestinal mucosa, are the more volatile fatty acids. That is, acetic, propionic, caprylic, butyric, etc. Lactic acid is non-irritating. Let us see what bearing these facts have upon the subject under discussion. If there were poured into the intestinal canal a solution rich in fats, and the fermentative processes, which under these circumstances might be quite active, were not absolutely normal, there would then be produced an excess of those irritating by-products of fermentation already mentioned, resulting in an increased flow of mucus from the mucosa and an increase of the blood-supply to the intestinal wall. In other words, a congestion would occur. Such congestion is ordinarily associated with increased action of the intestinal musculature. Were such increased action local, the result would be a spasm of the intestinal wall. This spasm is the essential feature in colic. The accumulation of gas is secondary, because in all fermentative processes gas is produced. But unless there is this spastic action of the intestinal wall incarcerating the gas within a segment of the intestine the pain of colic is not produced. This can be readily demonstrated clinically in children who pass large quantities of gas and yet have no colic.

We have, therefore, some definite indications for treatment of colic in these infants, and it will be interesting to note with what success such treatment may be followed. It is common experience that the simple expedient of reducing the quantity of breast milk, either by shortening the length of the nursing period or by lengthening the interval between feedings, or both, is sufficient in most cases of beginning colic to overcome the difficulty. But usually the physician is not called upon to look after a case of colic until the colic has developed so far that

simple measures are no longer sufficient. In these cases the reduction of the food is usually the first measure tried, but, as a rule, success does not follow such simple means.

There is another class of cases where reduction of breast milk is neither advisable nor possible, because in these cases, as in two of the cases here demonstrated, the quantity of breast milk is reduced rather than increased, and the colic can only be explained by the circumstance that where the quantity of breast milk is small the proportionate amount of fat is exceedingly high, a circumstance which tends to increase fermentation and reduce putrefaction.

It would seem that in this latter class of cases the indications for treatment consist in attempts to increase putrefaction and to direct fermentation in the bowel along normal channels. With this in mind it has seemed logical to me to give these children casein before nursing for the purpose of increasing the putrefaction, and cultures of the lactic acid bacillus in order to stimulate fermentation of the non-irritating kind.

Let us take each one of these cases now and find out what measures were taken to overcome the colic and the results of these. In the first case the child was ordered to be put to the breast for ten minutes every four hours. After the nursing the breast was to be pumped. Having determined the quantity of breast milk taken at each nursing, that obtained by pump was to be skimmed and the total quantity made up to $3\frac{1}{2}$ ounces. In addition to these measures, 1 gram of powered casein was given before each feeding, and one-third of a tube of a liquid culture of the lactic acid bacillus morning and evening. I might state here that it is extremely hard to obtain good powdered casein, and that for the purpose one may curd skimmed milk. Usually the curd from 1 ounce is about the quantity to be used before each nursing.

Let us see what the result of this rather intensive treatment has been. In the first case, two days after the institution of this treatment the mother reported that the child had gained 3 ounces, and that, while there was much gas and some spitting, there was no colic and that the child had slept all night. In the

four days following the child had gained $3\frac{1}{4}$ ounces more, and colic had remained practically absent.

In the second case the mother was advised to give the child 1 gram of powdered casein previous to nursing. On the next morning the mother reported that the colic still persisted and that the child had obtained 20 ounces from the breast. She was advised to pump the breast, skim the breast milk, and give the child 3 ounces of skimmed breast milk every four hours. The casein was to be continued and, in addition, one-third of a tube of the culture of lactic acid bacillus was to be given morning and evening. The next day the mother reported no results. She was then advised to nurse the baby for five minutes and pump afterward, proceeding as before. After this colic was absent for five days, since which time there has been slight colic, but this has been so little that it has been practically disregarded by the mother. When last heard from, on the 20th of last January, there was no colic. The child had been gaining steadily, the weight having advanced to 9 pounds, $7\frac{1}{2}$ ounces. The treatment was continued, but it is altogether likely that by this time the mother has seen fit to stop these measures.

In the third case, instead of the artificial food mixture which had been given previous to the time when I saw the child, I advised, contrary to all rules, that the breast milk be supplemented with albumen milk, a mixture as follows being used:

Albumen milk	18 ounces.
Dextrimaltose	$\frac{3}{4}$ ounce.

Sufficient of this mixture was given to make the single feeding up to 3 ounces. The result was quite amazing. The colic disappeared as if by magic and did not return. The weight increased and the child went along beautifully. This child has continued in good shape until the breast milk was reduced so low that I felt it wise to take it off the breast entirely and put it on an albumen-milk mixture. Almost immediately the child developed a constipation and, as is my usual custom, I substituted some dry malt soup extract for some of the dextri-

maltose. The quantity used was not sufficient at first, so I increased it further, with the result that the quantity of gas which the child was passing increased, colic developed, and the number of stools increased from one to five in twenty-four hours. This condition readily responded to treatment, however, which consisted of reduction of the malt soup and increase of the dextrimaltose. In my experience colic in artificially fed infants has been more frequent in those fed on malt soup than where any one other single article of food could be suspected.

If, now, we consider these cases together we see that in all of them the results depended almost entirely upon the thoroughness with which we carried out our treatment. In the second case, where we proceeded slowly, the reaction was slow, but in this case there was a factor which entered in which must always be taken into consideration, and that is the length of time since the colic had first been noticed. Both the first and second cases were as severe cases of colic as one is likely to meet with, and therefore I feel that they may be regarded as rather conclusive tests of the efficacy of the treatment.

Before this clinic is closed I want to point out another circumstance which surrounds these 3 cases. You will notice that no two of them have been treated in exactly the same manner. That while the principles of treatment were the same, their application was somewhat different. It seems to me that it is a mistake to try to teach you how to apply principles; that is one of the things which you must learn yourself. I can, however, lay down these principles and show you how to apply them. No two cases are the same and, therefore, no two cases can be treated in exactly the same manner.

One thing more, colic is a condition which in all likelihood has caused sleepless nights for untold generations. Such conditions do not go without their attempts at remedy. Such attempts have nearly always been directed against the pain and not against its *cause*. It is a very simple thing to prescribe some anodyne or opiate for these infants and go placidly on your way, assured that whether the child is benefited or not the family are pacified. This, however, is not the practice of medicine, and one is only

justified in giving sedatives to these children when every other means has failed, nor must he think that by using such methods he has done any more than temporarily allay the pain. The deep, underlying cause of the colic is still there, and the condition will again manifest itself as soon as the soothing syrup is withdrawn.

CLINIC OF DR. PETER BASSOE

PRESBYTERIAN HOSPITAL

ABSCESS OF THE BRAIN

Cases Illustrating Various Forms and Their Etiology.

I. TEMPORAL LOBE ABSCESS IN CONNECTION WITH MIDDLE-EAR AND MASTOID SUPPURATION

CASE I.—This patient, a domestic fifty-three years old, tells us that she has had a discharge from the right ear for several years. Nearly two years ago she was admitted to another hospital suffering from severe headache. She was irrational and confused and had difficulty in understanding what was said to her. The leukocyte count was 12,000. A radical mastoid operation was performed, and when the patient was seen in the evening of the day of operation she was clear mentally and had no speech disturbance. However, a slight optic neuritis was present, but the pupils and visual fields were normal. There were slight weaknesses of the lower part of the face on the left side and of the left arm and leg. The tendon reflexes were increased on the left side, where also an ankle-clonus and Babinski sign were obtained. Sensation to pain was diminished on the whole left side. These symptoms strongly suggested an abscess in the right temporal lobe, and a month later a trephine opening was made, but no pus was obtained on aspirating the temporal lobe. When the patient was seen six months later she was clear mentally, but the impairment of motion and sensation on the left side persisted and, in addition, she had developed a left homonymous hemianopsia. Lumbar puncture was made and the results showed that a certain amount of meningitis was present,

as the fluid was cloudy with a yellowish tinge and gave a cell count of 770. The diagnosis of temporal lobe abscess was still insisted on, and this time the surgeon succeeded in evacuating a considerable amount of very thick pus. It is probable that the suppurative process was present at the time of the first operation, but the pus may then have been too thick to be aspirated. Streptococcus was obtained in cultures. Considerable headache persisted and there was great difficulty in maintaining satisfactory drainage.

Half a year ago she had two convulsive seizures, but, on the whole, she has gradually improved. The drainage-tube was removed three weeks ago. As you see, she still presents signs of a partial left hemiplegia. The tongue on protrusion deviates to the left and the grip is weaker. There is no longer any distinct impairment of sensation and, what is very interesting, the hemianopsia no longer exists. It may be assumed that the hemianopsia was caused by pressure from the abscess and, perhaps, by cerebral edema in the vicinity of the abscess which, being located in the temporal lobe, was close enough to the optic radiation carrying the visual fibers forward from the occipital lobe to cause a temporary hemianopsia. Similarly, the partial hemiplegia and the temporary hemianalgesia must have been caused by pressure upward in the direction of the motor and sensory tracts.

Altogether the patient must be congratulated on the outcome, and it is very fortunate for her that the abscess was located in one of the so-called silent areas of the brain. If the left temporal lobe had been affected, she almost certainly would have had a marked sensory aphasia, and this would have been permanent, as a large part of the temporal lobe has been destroyed.

We will now present another middle-aged woman in whom the condition probably is very similar, but there is this important difference, that in this patient the left side of the brain is affected.

Case II.—The patient is a married woman, fifty-six years old, who has been the victim of delusions of persecution and jealousy for at least eighteen years, and has been a follower successively

of various peculiar religious cults. It is, therefore, not surprising that when two weeks ago she began to use wrong words and complain of headache, both her physician and her family thought that she merely had an aggravation of her mental trouble. It is, however, obvious that a real aphasia is present. She has great difficulty in naming common objects. She cannot understand anything that is at all complicated, and while she sees printed and written words, they have no meaning to her—*i. e.*, she has complete alexia. As she is right handed we may conclude that she has a lesion affecting the auditory speech center in the left temporal lobe and the "reading center" in the angular gyrus more posteriorly. On further examination we note that the lower part of the face is weaker on the right side, that the tongue when protruded deviates to the right, and that while there is no distinct weakness in the right arm and leg, the tendon reflexes are exaggerated on the right side and the abdominal reflex is absent on the right side. The plantar reflexes are normal and no Babinski sign is obtained. This is the rule when the motor function of the leg is unaffected. Furthermore, on ophthalmoscopic examination we find moderate choked disk on both sides, more marked in the left disk, where a few hemorrhages are seen. We can, therefore, conclude that the acute brain lesion in the temporal lobe is associated with increased intracranial pressure. On further questioning we learn from the family that the patient had a cold five weeks ago, that she complained of pain in the left ear, that the drum ruptured, and that there was a discharge of pus for a few days. This makes the chain of evidence complete: we make a diagnosis of acute abscess of the left temporal lobe. And applying the lesson from the previous case we may hope that the more distant symptoms, such as the alexia and papillitis, may disappear if the abscess is drained, but some degree of auditory aphasia is likely to remain. There is no hemianopsia and no anesthesia. There is apparently only a minimal meningeal involvement, as the spinal fluid is clear, with a cell count of 10, but a little globulin is present. The blood gives a leukocyte count of only 8000. We will advise trephining over the temporal lobe and exploration with an

aspirating needle. If pus is found, drainage should be established.[1]

II. TRAUMATIC BRAIN ABSCESS

Case III.—A female child seventeen months old was admitted to the service of Dr. Dean Lewis on May 17, 1917. Two weeks previously a piece of stove-pipe had fallen on her head, causing a bleeding wound on the top of the head 1 cm. to the right of the median line and 1 cm. behind a line connecting the right and left

Fig. 256.—Drained recut abscess of parietal lobe. Note dilated ventricle on opposite side.

auditory meatus. One week later pus appeared and the child became "restless and feverish." On admittance, examination, aside from the discharging wound, was practically negative. There was no rigidity of the neck or Kernig sign. *x*-Ray examination showed a small circular opening in the skull at the point mentioned. The leukocyte count was 9900 on May 17th and 10,900 on May 21st. The temperature ranged from 98° to 101° F.

[1] This advice was not taken and the patient died some weeks later.

and the pulse-rate from 100 to 120, until an operation was performed on May 23d, when the bone was exposed at the place of injury, where a defect 8 mm. in diameter was found. Here the dura protruded, and when an aspirating needle was inserted 3 drams of yellow pus were withdrawn. It was then incised and a rubber drain inserted. Pure cultures of the Staphylococcus albus were obtained from the pus. The fever, however, persisted, and the temperature on June 11th reached 103.2° F. On the next day an aspirating needle was inserted and ½ ounce of pus was obtained. Drainage-tubes were introduced. The irregular fever persisted, however, and the child gradually failed and died on July 29th.

The abscess cavity was found to have extended far beyond the site of the injury, as it reached backward in the upper part of the parietal and occipital lobes almost to the occipital pole. In fact, it reached its largest dimension in the occipital pole. It extended anteriorly a short distance into the frontal lobe.

In Fig. 256 is seen the appearance of the cross-section of the brain near the site of injury and subsequent drainage.

Case IV.—A man twenty-one years old was admitted to the service of Dr. Bevan in the hospital in a semiconscious condition, moaning with pain. He had been in his usual good health until the day of admittance, when he suddenly developed severe headache. It was noted that he had a discharging sinus in the right frontal region. His temperature was 100.4° F., pulse 50, respiration 18. Leukocyte count was 14,900. There was bilateral choked disk, more marked on the right side. The reflexes were normal and there was no paralysis. It was learned that the discharging sinus had existed since he received a gunshot wound in the right frontal region eight years previously. It was said that the bullet had penetrated the skull, but had not entered the cranial cavity, and that it had been removed by a physician. The patient died less than twelve hours after admittance.

The necropsy revealed a huge abscess which occupied the anterior half of the right hemisphere. The dura was adherent both anteriorly and externally, and beneath the latter point of

adhesion a flattened piece of bullet was seen (Fig. 257). It will also be noted that a second and smaller abscess cavity was present above the large one. A very dense wall surrounded the whole abscess.

It is curious that the man could have carried this enormous pus collection for years, and had his whole right frontal lobe completely excavated, and yet never suffered from any focal symptoms. It is equally curious that the symptoms of increased

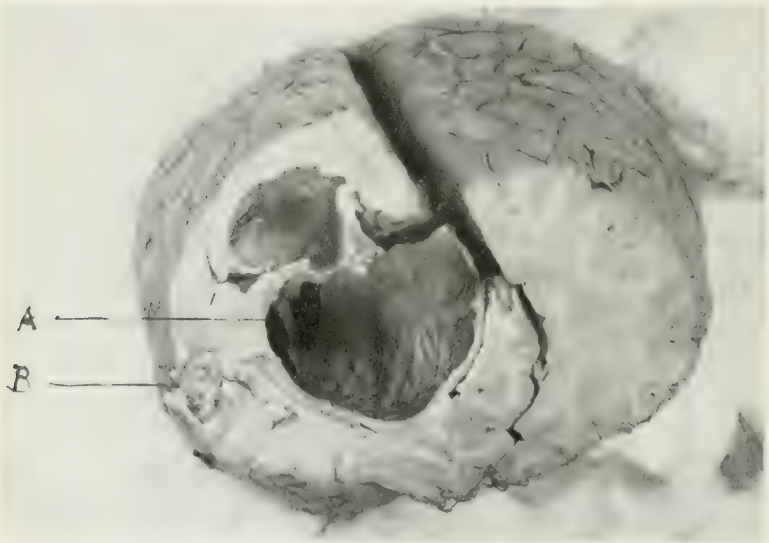

Fig. 257.—Old abscess of right frontal lobe: A, Abscess; B, fragment of bullet.

intracranial pressure should have appeared with such suddenness and severity.

The following case is of great interest, as it shows the difficulty in locating the principal brain injury, and that mistakes may be made even when the location of the skull fracture and the only focal symptoms with apparent conclusiveness place the lesion in a certain part of the brain.

Case V.—A man twenty-nine years old was seen with Dr. W. R. Parkes at the Evanston Hospital where he had been

admitted May 13, 1917, immediately after having been struck by an automobile. There was a contused wound in the left temporal region and there was hemorrhage from the nose and ears. The pulse-rate ranged between 48 and 60 and the respirations were slow (13). On June 14th lumbar puncture yielded a bloody fluid. The patient remained in a stuporous condition, with a temperature ranging from 99° to 101° F. A fracture involving the left temporal bone was demonstrated.

When examined on June 20th he was still very dull and confused and his speech was indistinct. There was slight weakness in the lower facial muscles on the right side, the right grip was weaker than the left, and there was slight ankle-clonus on the right side. The other reflexes were equal and normal. As these symptoms indicated a lesion of the left side of the brain, operation at the site of injury to the skull was advised. On June 26th Dr. C. J. Swan operated in the left temporal region and found the dura tense. After the dura was opened the brain did not pulsate; ½ inch beneath the surface there was a cavity which contained about an ounce of clear fluid, and after its evacuation the brain pulsated normally. The patient improved and went home. He soon became disturbed mentally, was violent, and had to be removed to the Chicago State Hospital, where later signs of meningitis developed. On September 26th lumbar puncture yielded a cloudy fluid with cell count of 1541 and negative Wassermann test. The patient died on September 27th and necropsy by the coroner's physician revealed a purulent meningitis, and an abscess at the tip of the right frontal lobe. A few months later the brain was received in formalin for further study. It was in bad condition, as it had been incised when fresh.

The following changes could be made out: 1. A fibrinous exudate on the under surface of the pons, medulla, and cerebellum. 2. Rusty discoloration and soft consistency of the under surface of the right frontal lobe, the necrosis extending to a depth of 15 mm. on the inferior surface and reaching almost to the tip of the lobe. The necrotic area measures 6 cm. anteroposteriorly and in its center a little pus is seen. 3. A rust-colored hemorrhagic and softened area on the under surface of

the left frontal lobe. 4. An area of superficial necrosis with denudation of the pia at the site of operation in the left temporal lobe.

It is clear that the direct injury on the left side of the brain was comparatively slight, but as it involved a highly differentiated functional area it produced focal symptoms. The really important injury occurred in the right frontal lobe at the site of the contrecoup. Subsequently this part of the brain underwent necrosis, and finally micro-organisms, probably brought by the blood-stream, produced an abscess here, which ruptured and resulted in the fatal meningitis. The production of brain abscess in this manner at the site of the contrecoup is unusual, but is recognized as possible. Ordinarily traumatic brain abscess is produced in one of the following three ways: 1. Infectious material is introduced directly into the brain at the site of the cranial injury, as in our Case III. 2. In cases of basal fracture bacteria may reach the brain from the nose or pharynx. 3. Bacteria may reach the brain by way of infected thrombi in the emissary veins at the site of injury.

III. BRAIN ABSCESS SECONDARY TO LUNG ABSCESS

Case VI. A woman thirty-six years old had a tonsillectomy under ether in July, 1916. The tonsils contained considerable pus. Two months later she had pulmonary hemorrhage followed by expectoration of foul green sputum. There was dulness over the lower part of the right lung, and on October 7th a lung abscess was drained. The wound closed in January, 1917. Later, cough and expectoration grew worse again, but when the wound was reopened only blood was obtained. In September, 1917 the patient developed severe right-sided frontal headache and morning vomiting. On September 19th she became stuporous. She was readmitted to the hospital. There was a bilateral choked disk, but no other neurologic findings were noted until September 24th, when a bilateral Babinski sign was obtained. The spinal fluid gave a negative Wassermann test, a cell count of 957, a positive globulin test, and positive Lange gold test (1-2-3-3-3-2-1-0-0-0). Unfortunately, a weakly positive Wasser-

mann test was obtained with the blood, which caused some hesitation in making a diagnosis of brain abscess, which otherwise seemed the only possible one. The leukocyte count in the blood was 17,050. At the necropsy several small empty abscess cavities in the lower lobe of the right lung were discovered, as well as firm pleural adhesions.

The brain showed the right hemisphere in the anterior portion to be decidedly larger than the left, and on section we see an abscess cavity, the anterior end of which is near the right frontal pole. It occupies the lower third of the right frontal lobe and extends along the outside of the olfactory bulb in the inferior portion of the frontal lobe to a point about 1 cm. behind the level of the tip of the temporal lobe. It has the appearance of an acute abscess, as it has no distinct wall such as we saw it exquisitely in the brain of Case IV.

This case is an illustration of the class of brain abscesses which ranks in frequency next to the traumatic and otitic ones, namely, brain abscess secondary to suppurative disease in the lung, notably lung abscess and bronchiectasis. In this case the lung abscess, in its turn, had followed tonsillectomy under ether anesthesia, and it should teach us that this procedure is not without danger.

IV. CEREBELLAR ABSCESS AND ABSCESS OF SPHENOID SINUS

Case VII.—A girl thirteen years old was first seen at her home on April 5, 1919, with Dr. John Roberts, of Portage, Wisconsin. Three weeks previously she had complained of headache, digestive disturbance, and photophobia. The headache grew more severe and she vomited frequently. A blood count on March 20th had shown 9600 leukocytes. The pulse-rate was usually 60. The temperature did not exceed 99.8° F. From the beginning of the illness she was very drowsy and slept much of the time. Spinal fluid obtained on April 1st gave a negative Wassermann test, positive globulin test, and a cell count of 31 lymphocytes. Her previous health had been good. The tonsils had been removed four years previously and since that time no trouble with the nose or throat had been noted. Examination

on April 3d revealed a bilateral choked disk of slight degree. The reflexes were normal and there was no paralysis and no stiffness of the neck. She was brought to the Presbyterian Hospital four days later, and on admittance the photophobia was very marked, the pupils were dilated, and there was paresis of the left external rectus; hearing good, no discharge from the ears. The reflexes were normal with the exception of the abdominal, which were weaker on the left side than on the right. Roentgenologic examination of the head was negative. The leukocyte count on April 8th was 10,400. The temperature ranged from 98.6°

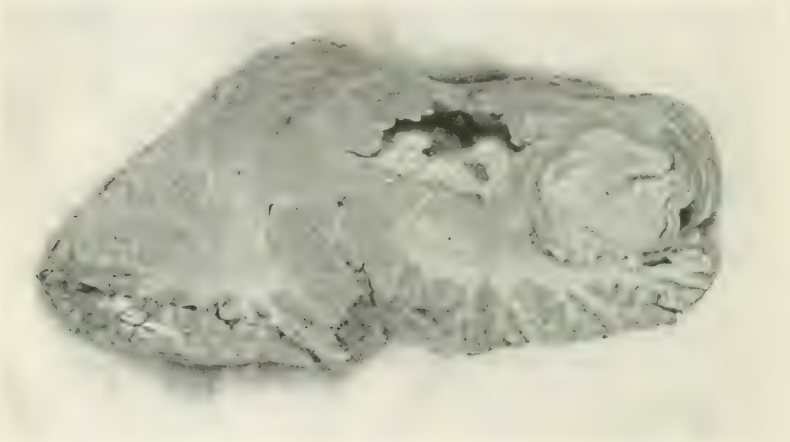

Fig. 258.—Acute abscess of cerebellum.

to 100° F. A tentative diagnosis of brain tumor was made, and in the absence of localizing signs no radical operation was advised. On April 10th a subtemporal decompression was made by Dr. Carl B. Davis. The brain pressure was extremely high. The patient had some relief, which lasted for two days only. Then the headache again became very severe and the swelling of the disks increased. The temperature reached 101° F. and the pulse-rate, which had ranged between 80 and 100, became more frequent. On April 18th, under local anesthesia, the left lateral ventricle was punctured and 3 ounces of clear fluid removed. This fluid gave a cell count of 11 and a very weak globulin test.

The Lange gold test was practically negative. This procedure gave considerable relief for a few days, but the leukocyte count rose to 14,000 on April 21st and 16,400 on April 23d. Death occurred on April 24th.

At the necropsy, which was held by Dr. B. O. Raulston, an abscess was found in the left lobe of the cerebellum in its upper and anterior portion (Fig. 258). The right sphenoid and posterior ethmoid sinuses contained a large quantity of mucopus which, however, was not under high pressure. The venous sinuses were normal and there was no pus in either tympanum. A staphylococcus was obtained in cultures from the cerebellar abscess.

CLINIC OF DR. ROBERT SONNENSCHEIN

POST-GRADUATE MEDICAL SCHOOL

SOME NON-SUPPURATIVE FORMS OF HEADACHE

March 16, 1920.

No pains, it seems to me, are more frequently complained of by our patients than headaches, in one form or another. This distress is not a disease, but only a symptom, whose cause may lie in disturbances of many parts of the body apart from the head. Thus, while many conditions, such as the anemias, cardiac and renal lesions, intestinal or other intoxications, etc., may produce headache, we are today occupying our time with cases in which some lesion or abnormality within the head and particularly the nose is the apparent causative factor in the production of the pain.

The sensation we call headache must be some irritation or affection of the fifth cranial nerve (trigeminus) either by contact or pressure directly along the fibers of this sensory nerve, by way of the circulation, or reflexly by communication with other nerves, such as the sympathetic.

A number of the patients whom we will present to you have been under observation for several weeks or months, and one of them for several years. Today we will be able to see the effects of treatment, or at least to summarize our observations regarding the various individuals and their symptoms. Headaches are, of course, caused by many inflammatory or infectious processes in the ear, nose, or throat, perhaps most often by acute or chronic sinusitis. The cases we will present to you this afternoon, however, are all of a non-suppurative type, some of them being merely mechanical in origin or character.

The first 2 cases are those of young women with practically the same symptoms and findings.

Case I.—Mrs. R. P. F., aged thirty-four, a housewife, came here a few months ago complaining of headaches that had been present almost continuously for six weeks. These pains were noted chiefly in the left frontal and vertex regions. The patient was first examined by an oculist, who found some error of refraction which was corrected with glasses, but he stated at the time that he did not think the condition of the eyes was in any way responsible for the headaches, and this was demonstrated by the continuance of the pain after the use of glasses. The examination of the nose at that time showed a deviation of the septum to the right, with contact of the middle turbinates. Transillumination gave a shadow over the right maxillary antrum, but on further investigation no pus was found. There was considerable tenderness over the inner half of the floor of the frontal sinus, the so-called Ewing's point. On cocainizing the left middle meatus and then opening it the patient had considerable relief. The left middle turbinate was then infracted and pressed away from the lateral nasal wall. The patient returned a week later and stated that the headaches had been practically absent. Today she is again here and, as you have heard, states that she has been entirely free from the headache which formerly distressed her greatly. Do not let me forget to state that she was given a prescription containing an organic silver preparation to use as an astringent in her nose after the middle turbinate had been dislocated, in the manner previously mentioned.

Case II.—Miss H. G., aged seventeen, a student, when first seen a month ago complained of dizziness and a feeling of faintness most of the time, but more so after using the eyes, which became considerably irritated after reading. She had moderate bilateral frontal headaches, worse during the morning hours, and, as she expressed it, "felt very nervous." There is a history of frequent head colds which clear up easily, but there has otherwise been no nasal discharge, nor any obstruction to breathing.

At the first examination the nose showed large middle turbinates, especially the right, lying very close to the lateral wall,

so that the entrance to each middle meatus was practically occluded. No abnormal secretion could be seen. There was marked tenderness of Ewing's point on each side. The pharynx presented submerged tonsils, but no pus or caseous plugs could be expressed from the crypts. Transillumination of the nasal accessory sinuses was negative, but *x*-ray pictures made at the same time showed a very slight cloudiness of the frontal sinuses. The latter findings may be discounted, we believe, in view of the fact that transillumination was negative, that the patient had never observed any pus issuing from the nose, and that, after cocainizing the middle meati and using suction, we were unable to obtain any pus or even thick mucus. Here we have an instance, often noted, where the *x*-ray and transillumination do not agree, and where we must rely on the clinical findings. While we know that in the various branches of medicine laboratory and other tests are often of the very greatest aid and value, we must remember that careful examination and weighing of the clinical evidence is the most important factor in making a diagnosis. If the tests, such as those reported from a laboratory, or derived by means of tuning-forks, etc., confirm our clinical findings, they help greatly, but if at variance with the symptoms and signs noted, they must be "taken with a grain of salt." In this particular case the cloudiness of the frontal sinuses was uniform and symmetric on the two sides, and was probably due either to a moderate congestion or thickening of the mucosa, or possibly to an unusually thick anterior wall.

When the middle meati were swabbed by means of applicators moistened with a 10 per cent. solution of cocain, the mucosa shrunk and the meati thus opened, the patient, to whom no suggestion had been made, at once said her "eyes felt more comfortable." Guided by this phenomenon, an endeavor was made to permanently open the meati by infracting the middle turbinates and pressing them away from each lateral wall and toward the septum. At the same time the patient was given a prescription for an astringent mixture to be applied in the region of the middle meati.

On returning to us a week later she reported that the head-

aches and eye fatigue had greatly improved. At present there is entire absence of all pains and distress.

How shall we explain these 2 cases and the method of their relief? It was Ewing and Sluder who, quite a number of years ago, described a condition in which headaches occurred which were formerly ascribed to some trouble in the eyes, but, in reality, due to nasal disturbances accompanied by ocular symptoms. Either because of an unusual approximation of the uncinate process to the bulla ethmoidalis, or of the middle turbinate to both these structures, the infundibulum is practically closed, especially when any inflammatory swelling of the mucosa occurs. There is, then, a diminution in the air pressure within the frontal sinus and we get the so-called "vacuum headache." This is usually a constant pain with varying exacerbations, usually worse in the mornings, and often causing inability to use the eyes for any close work. Sometimes there is actual pain on moving the eyes from side to side or up and down. Characteristic of this form of headache is the great tenderness found on making pressure against the inner and upper wall of the orbit; that is, the orbital plate of the frontal bone in its nasal half, the os planus of the ethmoid, and especially the region of the pulley of the superior oblique muscle.

Relief may often be afforded merely by the use of astringents in the region of the middle meatus; in other cases infraction of the middle turbinate and pressing it toward the median line of the nose is required; and in some instances removal of the anterior tip of the middle turbinate may be necessary, especially if it is hypertrophied or bulbous. In both of these patients, as you have seen, infraction of the middle turbinate and the use of a mild astringent gave complete relief. Here, as in all nasal work, conservatism should be the keynote of our efforts; never sacrifice needlessly any of the structures whose mucosa bears the precious columnar ciliated epithelium, for the scar which forms is covered by a flat, squamous epithelium which does not serve the same important function as the former.

Case III.—This patient presents headaches showing some of the same symptoms as the two we have just discussed, but the

mechanical factor in the production of the headaches is different in that we have a pressure producing the pain and not a vacuum. Miss M. R., aged twenty-three, a stenographer, consulted us six months ago and gave the history at that time of having had for several years, and especially during the previous two months, severe right supra-orbital headaches, worse in the mornings. There had been no abnormal nose or throat discharge for the past nine years, but prior to that time she had for two weeks a yellow, purulent discharge. At no time has there been any nasal obstruction. During the two months before she first came to us she had had at least five or six very severe attacks of headache, accompanied by nausea and vomiting, and necessitating rest in bed for a day or so, but almost every day there had been some headache, as above described.

Examination of the nose revealed large tubercular septi, with contact especially of the right middle turbinate body. Transillumination was negative. After shrinking the mucosa of the right middle meatus and applying suction no pus or abnormal secretion was obtained. The pharynx showed flat, submerged tonsils, but no pus in the crypts. Trichloracetic acid was then applied to the right tuberculum septi. The patient returned in a few days and stated that some relief had been obtained. Guided by the fact that the moderate shrinking of the tissues had relieved the pressure exerted by the right middle turbinate upon the septum, the parts were thoroughly cocainized, and the turbinate then infracted and pressed away from the septum and toward the lateral nasal wall. The patient on coming back a week later stated that she felt entirely free from headaches. Today she tells us that in the past five months she has had only on very rare occasions slight twinges of pain, but that none of the severe headaches or distress which she formerly experienced have been present until within the last two days. Today we see that the right middle turbinate has apparently sprung back to its original position and is pressing upon the septum. We will again push it away from the latter, and if the patient is then not permanently relieved, it will be necessary to remove a portion of the anterior end of this middle turbinate.

In this case we see that we have headache caused by an entirely different condition from that present in the first 2 cases. In the latter the middle turbinate body, while lying close to the lateral nasal wall, produced a closure of the middle meatus, with consequent rarefaction of the air in the frontal sinus; in the patient now under discussion there was pressure of the middle turbinate body on the tuberculum septi—that thick portion of the septum at the junction of its upper and middle third. This region has a very vascular mucosa; variations in the amount of venous engorgement in the mucosa covering this structure and the anterior end of the middle turbinate body, depending upon such factors as posture (head lying low, as in sleep), local irritations or infections, traumatism in the nose, etc., may cause contact, pressure, and pain. The headache may be felt in different parts, such as the forehead, the root of the nose, in or behind the eyes, in the vertex, or at times even in the occiput.

The treatment is directed toward removing the contact of the middle turbinate and the septum. This may be accomplished at times by non-operative, or in other cases only by mechanical or operative measures. If venous engorgement is an etiologic factor, free catharsis, treatment of any cardiac lesion if present, and avoidance of low position of the head during sleep or work, should be advised. If marked intumescence is present, and fails to yield to simple measures such as these, cauterization of the mucosa with trichloracetic acid or the galvanocautery is often of great benefit. Care must be used first to avoid destroying any more of the overlying mucosa than is absolutely necessary by merely making a linear cauterization, and second, to prevent consequent adhesions between turbinate and septum. This may be accomplished by means of petrolatum applied between the two structures or, better still, by placing a thin piece of dental wax between the two surfaces and retaining it in position for a few days, just as has been my custom after cauterization of the inferior turbinates. Should the headaches not be relieved in this manner, we can very easily infract the middle turbinate and press it away from the septum, as was done in the case just before us. This is very easily and quickly accomplished without

danger or delay to the patient, and without pain if proper cocainization is carried out. Should the turbinate later spring back in place, as happened to us in this patient, and again produce pressure, we may repeat the infraction, and if this also fails to prevent recurrence of contact and pressure, or if the space between the septum and lateral nasal wall is so narrow that it is impossible to move the middle turbinate, it is advisable to snare off just enough of the anterior tip of the turbinate to prevent further contact. If the septum is badly deviated, one may avoid the removal of any turbinate tissue by performing a submucous septal resection.

These cases are often among the most gratifying ones met with in rhinologic practice, because a very annoying and at times exceedingly persistent headache is often quickly and either for a long time or permanently relieved.

Our next 2 cases will show headaches caused largely by the mechanical action of misplaced last molar teeth, so-called impacted molars.

Case IV.—Mrs. F. T., aged thirty-eight, a housewife, first consulted us six years ago, and at that time stated that she had been having, for eight months previously, severe pains of neuralgic character radiating from the right supra-orbital region across the temporal region, to the mastoid, and from there to the base of the skull. These pains were accompanied by vertigo and nausea and would last from one to four days. At no time had the patient had any purulent or other abnormal nasal discharge.

Examination of the nose showed a marked septal crest on the left side; also a rather large right middle turbinate body; some hypertrophy of the right inferior turbinate. In the pharynx some injection was present, but no other findings. Transillumination was negative. Despite this fact the right maxillary antrum was punctured and found free from pus or any other secretion. *x*-Ray examination showed that the right lower last molar was completely impacted and pointing toward the inferior dental foramen. The patient had also complained that at the time of the headaches vertigo was present, as we mentioned

before. In order to be sure that no labyrinthine involvement was present, the vestibular apparatus was examined and found normal; then a complete tuning-fork functional test was made and the hearing shown to be unimpaired.

The patient was then referred to an extracting dentist, who removed the offending molar. When she returned to us a few weeks later the headaches had disappeared. Now, after six years, she has returned because of pains in the right ear, and we find that the right drum membrane is considerably reddened. This condition, of course, has nothing to do with her former complaint, but is merely an acute otitis media. We are very glad to learn from her that she has had no recurrence of the headaches which were present before the impacted molar was extracted.

Case V.—Mrs. S. S. S., aged twenty-six, housewife, first consulted us a few weeks ago. She stated that for four years she had been suffering from pains in the left side of the face which radiated into the neck and the left ear. Examination of the nose showed some pus in the right middle meatus, and the source of this was demonstrated by transillumination, which revealed a shadow over the region of the right maxillary antrum. The nasopharynx was negative and the pharynx showed submerged tonsils with large caseous masses in the crypts, especially on the left side. The antrum was treated a few times and the pus disappeared. The pains in the left side of the face and neck, however, continued as severely as before. An *x*-ray picture showed that the left lower third molar was impacted. This was then ordered removed, and the patient returned shortly thereafter and assured us that the pains had entirely disappeared.

She is present today in order to have an infection in her right ear treated which has no connection with her former condition. This patient showed none of the vertigo or other labyrinthine symptoms noted in the case we demonstrated just before. All she complained of was the pain.

We have here 2 interesting cases in which severe headaches were caused by an apparently very simple condition, the impaction of a tooth. In the first case one would expect to find

a labyrinthine lesion because of the vertigo and nausea, and the fact that sudden movements of the head initiated the onset of these symptoms. Complete functional testing of the ear, however, showed no abnormalities. Even though transillumination of the nasal accessory sinuses was negative, the fact that the pains were on the right side led us to irrigate the right maxillary sinus, but no pus was found. The removal of the impacted molar completely relieved the patient.

In the second of these cases all labyrinthine symptoms were absent and the patient's pains were on her left side. At the same time she had an infection of the right maxillary sinus which promptly yielded to treatment. The pains on the other side of her head, however, were not relieved until the impacted third lower left molar was removed.

We thus see how a condition producing irritation along one small portion of the many branches and subdivisions of the fifth cranial nerve may cause pains in different portions of the head by reflection along the various parts of this great sensory nerve. Look, therefore, carefully in the mouth or throat as well as the nose for the possible source of a headache; in some cases transillumination or, better still, the *x*-ray picture may be of great assistance. We know, for instance, that pains felt in the ears are frequently due to conditions in the teeth, pharynx, or larynx, without any tangible aural findings. In the same manner headaches may be present in the form of referred pains.

Case VI.—The last case which we will present this afternoon is one in which the most excruciating pain persisted for a long time and caused great distress, which incapacitated the patient for her usual household duties. Mrs. K. T., aged forty-six, first consulted me eight years ago. At that time her history was that for sixteen years she had suffered from very severe neuralgic pains in the left side of her face. These pains began in the teeth, root of the nose and left cheek, radiated to the mastoid and occipital region, and then down the left shoulder into the arm. There was some pain present every day, but at least twice a week the paroxysms were so intense that the patient was compelled to remain in bed. On the assumption that the "neuralgia" was

due to diseased teeth, all of the upper ones had been extracted, but without affording any relief whatever. The patient's nervous system after a time became exhausted from the constant and severe suffering. She also complained of a tinnitus aurium on the left side which appeared at times. No purulent or other abnormal nasal secretion was ever noticed by the patient, nor had she ever had symptoms in other parts of her body which might in any way have had any bearing on the headaches.

Examination of the nose showed a septal spur low down on the left side; the inferior turbinates were somewhat hypertrophied. The tonsils were large and contained many foul, caseous plugs. Transillumination of the sinuses was negative.

On introducing an applicator carrying a drop or two of 10 per cent. cocain solution back of the posterior end of the left middle turbinate a most frightful paroxysm of pain ensued. The eyes teared and the patient exclaimed that the pain she then felt was the one she had experienced for sixteen years. After a few moments the part which had been touched was desensitized by the cocain and the pains then entirely ceased, only to return after a day. On repeating the cocainization the pain disappeared and then a ½ of 1 per cent. formaldehyd solution was applied. At intervals of three or four days the nose was cocainized and increasing strength of formaldehyd solution, up to 2 per cent., applied just posterior to the middle turbinate body in the region of the sphenomaxillary fossa. After six or eight treatments the patient's pains entirely disappeared. During the course of the past five or six years she has occasionally returned and stated that the pain had recurred for a few days in a very mild form; two or three applications would then be made, with disappearance of all symptoms for many months. Today we see her because she has had trouble with her tonsils. Infection of her throat, together with pains in her joints, makes us feel that the tonsils may be the seat of a focal infection and that possibly a tonsillectomy is indicated. We are happy to hear from her lips that the headaches trouble her no longer.

In this case we have the history of a very severe headache, which during a period of sixteen years seriously incapacitated

the patient, and caused her to have all her upper teeth extracted on the strength of a mistaken diagnosis of neuralgia, supposedly of dental origin. She had, however, the typical pains caused by irritation of Meckel's or the sphenopalatine ganglion, namely, sharp pains beginning in the teeth, cheek, and root of the nose, radiating upward by way of the temporal region to the ear and mastoid process, then passing down into the pharynx, neck, shoulder, and even to the arm. Not all cases, of course, have so extensive a radiation of the pain. Our patient also showed a marked tearing of the eyes when a severe paroxysm was present.

Meckel's ganglion lies in the sphenomaxillary fossa and by means of its numerous communications sends sensory impulses to nose, orbit, nasopharynx, and palate. The diagnosis of involvement of this ganglion can usually be made by inserting an applicator just back of the posterior end of the middle turbinate body, passing slightly upward, backward and outward. If the ganglion is affected, and especially if it lies rather superficially, this contact of the instrument will cause severe pain, showing the characteristic radiation mentioned above. If the application of cocain in this region then causes the pain to disappear, the diagnosis is practically made.

The *treatment* of this very distressing condition suggested by Dr. Sluder is to inject a small amount of alcohol containing about 2 per cent. carbolic acid into the ganglion or its immediate vicinity. After thorough cocainization a long needle is inserted just behind the middle turbinate body and then pushed upward, backward, and outward until the bone covering the ganglion is penetrated; the syringe is then attached and the alcohol solution injected. If the ganglion or its immediate vicinity is reached, a most intense pain is caused, lasting from a few hours to several days. In these cases the consequent relief is much greater than where no pain or only a slight discomfort is produced by the injection, for in those instances it usually means that we have failed to reach the ganglion. Occasionally severe complications attending these injections have been noted by various men, such as severe hemorrhage, corneal ulceration, etc.

In our patient there was evidently a dehiscence in the bony

covering of the ganglion, for the slightest manipulation in this region caused a paroxysm which was quickly stopped by cocain. The condition was then relieved for many months at a time by applications of formaldehyd solutions without at any time employing a needle to puncture the bone. One of the saddest features about this case is the fact that all the upper teeth were needlessly sacrificed before we saw the patient and made the correct diagnosis.

CLINIC OF DR. RALPH C. HAMILL

WESLEY MEMORIAL HOSPITAL

THREE CASES OF CEREBROSPINAL SYPHILIS

THE first 2 of these cases are of interest largely because of the results that have been obtained while under treatment. In both of them the intraspinous method of medication has played a considerable part.

CASE I

The first case was seen first in December, 1915. He was forty years of age and was, in the eyes of the law, unmarried. The family history was of significance, in that one sister who had died of cancer had been insane. Five brothers and sisters were well. In the previous history the factors of significance were a chancre in 1912, moderately heavy drinking at various intervals, and considerable tobacco. Otherwise he had been a good workman, though it is perhaps relevant that he had never followed one line for any length of time.

When first seen by me he was in the Chicago Psychopathic Hospital, where he had been committed several days before, as insane. The history of this attack was to the effect that one month before he had a fainting spell which lasted a few minutes. About a week later he became very restless and nervous, was afraid that he was going crazy, and complained somewhat of headaches. Following this he had several attacks of excitement, and it was on account of one of these that he was committed. It was said by the attendants that at the time of the examination he was much quieter and more rational than upon entrance. However, he was boastful. He stated that whereas he "had been crazy, now he was all right, but all the rest were crazy." When asked where he was, he said, with a humorous expression, "I am in Cook County's Psychopathic Hospital, at least I

suppose it is Cook County's, but there are some fellows here who think they own it." He knows the day, the date, and about how long he has been in the hospital. In a humorous way he answers the query as to his civil state which, as above stated, was legally unmarried, but he was living with a woman who was supposed to be his landlady. He knew me for a doctor, the attendants as such, said he hoped I would be able to help him out of the fix he was in. Asked to subtract, mentally, 237 from 1000, he said, "37 plus 3 equals 40, that's 63, you'd have 763." There were no tremors about the face such as seen in the paretic, no tremors of the voice. "Around the Rough and Rugged Rock" and "Theophilus Thistle" were spoken correctly. There was no increased swaying as he closed his eyes in the test for static ataxia, no Romberg. The pupils were slightly sluggish in their reaction both to light and convergence. There was a bilateral optic neuritis. The neck was slightly stiff and there was a suggestion of a Koenig sign on either side. The deep reflexes were equal, but diminished. The superficial reflexes were normal. There was no sensory disturbance either superficial or deep.

During the examination he was very emotional, crying in a rather convulsive manner three times, once when questioned about his mother, once about his dead sister who was insane, and the other time when asked why he cried so easily. Lumbar puncture revealed fluid under increased pressure. There were 110 cells to the cubic millimeter. The Nonne, of course, was positive, Wassermann strongly positive, with 0.2 c.mm.

Comment.—There are two factors in this case which deserve some comment, namely, the signs of organic disease and the mental condition.

As to the former, the optic neuritis, the pleocytosis in the spinal fluid and its strongly positive Wassermann content and the signs of meningeal irritation, the stiff neck and the Koenig's; it is obvious that they represent an inflammatory condition of the meninges.

As to the latter, I believe the fact that a sister died insane is of significance, that is, I should say that these two children belonged to a family showing a markedly neurotic disposition which

was inclined to give way in the presence of organic disease, the sister dying of cancer after being insane for some time; the patient having a cerebrospinal syphilis with evidence of marked emotional instability, so unstable, in fact, that his family was led to commit him as insane.

Treatment.—In the treatment of syphilis in the insane or in people who for any reason are unable to adequately care for the cleanliness of their teeth it is very difficult to give mercury in the ordinary manner because, with beginning saturation, as soon as they have had a very few doses, gingivitis and salivation are set up. In this man the teeth and gums were in a very bad condition, hence it seemed very unwise to attempt to combat his syphilis with mercury. In December, 1915 it was very difficult to obtain salvarsan or neosalvarsan upon which one could depend, hence I attempted to use intraspinous mercurial treatment, *i. e.*, I wanted to put the mercury where in small amounts it would do the most good. I gave an injection of 10 c.c. of Parke Davis mercurialized horse-serum. This produced a very marked reaction, with considerable retraction of the head and a well-marked Koenig. This reaction lasted only twenty-four hours. Nine days later I gave him $\frac{1}{60}$ grain of bichlorid intraspinously dissolved in 12 c.c. of his own serum. This was repeated twice in the following month. One month later he was given $\frac{1}{110}$ grain of bichlorid in 10 c.c. of serum. At this time he was feeling very much better, had begun to gain weight, and the cell count in the spinal fluid was reduced to 12 per cubic millimeter. The insane symptoms had disappeared following the second injection, at which time the optic neuritis had also practically disappeared. He was given two more injections of $\frac{1}{120}$ grain of bichlorid dissolved in 20 c.c. of spinal fluid during the next month. By this time he was able to take good care of himself and so was put on mercury rubs. He rubbed $1\frac{1}{2}$ drams twice a day for two weeks and then once a day for six weeks. Six months later he again rubbed for six weeks. On January 22, 1917 the blood Wassermann was negative. In August, 1917 the spinal fluid showed but 2 cells per cubic millimeter and the Wassermann was frankly negative.

Present Condition.—The patient at present is doing very good work in a machine shop, of which he is foreman, and up until recently has enjoyed very good health. About a month ago he developed a breaking out of the skin of the face which looked like a well-developed herpes that had started to subside when I saw it. The blood was examined and a faintly positive Wassermann led me to recommend a course of salvarsan injections intravenously as preventive if not as curative.

Cranial nerves are normal, though the pupils, the small type, do not respond with wide movements when tested with light; reaction to convergence is also of small amplitude. Deep and superficial reflexes are normal. There is no subjective or objective sensory disturbance. The fact that the man has advanced from an ordinary machinist to a foreman in the shop speaks for his mental condition.

Comment.—Of course, no one can say that the man would not have recovered without treatment. However, he did recover with a very small amount of intraspinous medication, medication that produced local reaction, hyperemia of the meninges. We know that the meninges, like other serous membranes or membranes in general, are poorly supplied with blood. It has been a dictum of authorities for a long time that the pleura and the peritoneum become infected and seem to show but poor resistance thereto because of their nonvascular condition. Doubtless the meninges belong in the same class. When a syphilitic infection develops in the nonvascular meninges it has a chance to establish itself before the vascular reaction develops around it in an attempt to destroy it. It is my idea that an acute hyperemia of these relatively poorly vascularized tissues may act in a salutary measure upon the infecting organism.

CASE II

The second case is that of a woman first seen in the spring of 1916. Her father died at about fifty. He was considered "wild." Her mother was well at the age of sixty. The patient herself, an unusually well-developed woman of thirty, had had enlarged glands at birth and had developed a "sore" in the right

cervical triangle a few months after birth. She had considerable indigestion as a young girl and "typhoid" at thirteen, during which she developed a large sore over the left temporal region. She had had a great many headaches, occipital in character, lasting from one to two days for the past two years. They were quite severe, but were not associated with vomiting. About two weeks before I saw her she awoke one morning with difficulty of vision. This was due to a dilated pupil.

Examination showed the right pupil moderately dilated. It reacted but poorly both to light and convergence. There was the above-mentioned scar in the temporal region, otherwise there were no abnormal findings, The spinal fluid showed 100 cells per cubic millimeter, Wassermann was positive, with 0.4 c.c. She was put on mercury rubs, but in spite of these ten days later the left pupil suddenly dilated. It reacted poorly to light as well as to convergence. The rubs were then doubled in quantity, and during the next two months 70 drams in all were rubbed. The headache, which was the feature complained of most, gradually subsided. In two months she was given seven injections of neosalvarsan, then returned to two rubs a day for six weeks. She was then allowed a vacation of a month. I neglected to say that about a month before these rubs the spinal fluid showed 4 cells per cubic millimeter, the Wassermann positive with 1 c.c. of fluid. For the next six months she alternated between weekly injections of neosalvarsan and six-week periods of rubs, apparently getting along rather better on the rubs than on the injections. A rather severe headache coming on after a month's rest in July, 1917, she was given 0.75 neosalvarsan intravenously; 10 c.c. of spinal fluid were then withdrawn for examination and to make way for the expected increase of fluid, $\frac{1}{100}$ grain of bichlorid was dissolved in 20 c.c. of spinal fluid and then the spinal fluid reinjected. A headache was complained of toward the end of the withdrawal of the 20 c.c., which disappeared immediately upon starting the reinjection. The spinal fluid at this time showed 42 cells per cubic millimeter, the Wassermann positive with 0.2 c.c. In view of this strongly positive Wassermann test as well as the 42 cells, it was apparent that in

spite of the fairly intensive treatment with both mercury and arsenic the luetic process was still active. Therefore I determined upon a change in the treatment, namely, to give several intraspinous injections. Three weeks later the intravenous and intraspinous injections were repeated. The serum Wassermann was frankly negative, the spinal fluid showed no cells, the Wassermann was positive with 0.4 c.c. of spinal fluid. Four months were allowed to elapse, at the end of which time a lumbar puncture was done; the Wassermann was positive with 1 c.c. of spinal fluid, negative in smaller amounts, there were no cells. One month later, February, 1918, a spinal and intravenous treatment was given, which was repeated in three weeks. The fluid at this latter time was negative in the Wassermann test with 1 c.c. of spinal fluid.

A year then elapsed without treatment and without symptoms. About one week after my return to practice severe headaches reappeared, especially over the right eye. Examination showed that the right pupil was almost fixed to light, otherwise there were no pathologic findings. The spinal fluid, however, showed 20 cells per cubic millimeter and the Wassermann was positive with 0.4 c.c. Intravenous injection of 0.6 neosalvarsan and intraspinous of $\frac{1}{120}$ grain of bichlorid were given, and repeated in three weeks. The spinal fluid at this latter time showed but 2 cells and was Wassermann positive with 0.6 c.mm. Then at two-day intervals she was given 0.6, 0.45, 0.3, 0.3 neosalvarsan. Ten days elapsed and she was given 0.3 for three doses at two-day intervals. The spinal fluid then showed but 1 cell per cubic millimeter, the Wassermann was positive with 1.5 c.c., negative in smaller quantities. A period of three months was allowed to elapse. The spinal fluid then showed no cells and frankly negative Wassermann, even with 1.5 c.c. Again, four months later, similar negative findings were determined in the spinal fluid.

Comment.—I neglected to state that the husband had no knowledge of infection. His blood was Wassermann negative. It is possible that this unusually well-developed woman, who outside of the scar in the temporal region and teeth that were

somewhat irregular and perhaps a little shorter than normal, showed no developmental anomalies, was suffering from inherited lues. This history of the father's probable indiscretions, the enlarged glands at birth, the sore in the right cervical triangle, the "typhoid," with its large temporal ulcer, all speak for the possibility of the inherited lues. The failure of conception may also be considered as evidence bespeaking this condition. When I first saw her she had been married only four years. Certainly one is justified in considering this an unusually resistant case of luetic infection. It may be that one is also justified in considering the degree of resistance an evidence of the long-standing duration of the infection. In other words, the obstinacy may be due to the fact that the case is one of inherited lues.

I do not believe that the negative results in the spinal fluid could have been obtained with mercury, and I am not inclined to believe they could have been obtained with general systemic treatment. It is my conception that the intraspinous introduction of substances which can cause a marked reaction on the part of the meninges may, by virtue of this reaction rather than because of any inherently curative powers of their own, eradicate chronic inflammatory lesions. It has long been taught in the treatment of chronic conditions in the skin that the use of irritating substances may be necessary to stir up the defensive powers of the skin that they may oust the indolent infecting agent. At least that was the teaching some years ago. If it still holds true, it seems to me quite reasonable to consider any poorly vascularized tissue in the same light. Several times I have done a second puncture within twelve to twenty-four hours after an injection of $\frac{1}{120}$ grain of bichlorid dissolved in the individual's own spinal fluid. The spinal fluid at the time of this second puncture is that of a moderately severe meningitis, that is, it may contain anywhere up to 15,000 white cells. In the few cases in which I have done a third puncture three or four days after injection this marked pleocytosis has all disappeared, due, therefore, it must be, to a chemical irritation, no infection.

There are a few points relative to the technic of intraspinous treatment that might be seasonable. My method of procedure is as follows: The lumbar puncture is done with the patient in the lateral position, the needle passing to one side of the middle line in order to avoid the interspinous ligaments. I first withdraw from 6 to 8 c.c. for examination, then I put $\frac{1}{120}$ grain of bichlorid of mercury into a 20-c.c. Luer syringe, connect this with the spinal needle, and very slowly aspirate 20 to 30 c.c. of spinal fluid. As I am withdrawing the fluid I occasionally turn the syringe so that the bichlorid becomes thoroughly mixed throughout the mass of fluid. Occasionally the patient will complain of headache. At the beginning of the procedure I warn them that a headache may come on, and to be sure to tell me thereof. The headache will develop after the withdrawal of a certain number of cubic centimeters of spinal fluid. This means that the supporting influence of the spinal part of the cerebrospinal fluid is being too rapidly withdrawn and the brain is settling down into the foramen magnum. As soon as the headache is complained of I commence the reinjection without removing the syringe. This is done *very* slowly. I usually allow about ten minutes to reinject the fluid, the needle is then withdrawn, the patient cautioned to lie flat without more than one small pillow for twenty-four hours. He is allowed to go to the toilet, but otherwise he must stay down. An injection of $\frac{1}{4}$ to $\frac{3}{8}$ grain of morphin is ordered to be given four hours after the intraspinous injection, to be repeated in $\frac{1}{4}$-grain doses at two intervals of three hours thereafter, if necessary. I find that the pains come on at just about four hours after the injection and can be well controlled by this medication.

It is my practice to give a large dose of neosalvarsan intravenously at the same time that the intraspinous injection is made. It may be purely fancy, but I like to think that the blood that is going to the diseased meninges is as heavily loaded with antigermal substances as possible. In fact, I like to give six to eight neosalvarsan injections intravenously over a period of three to four weeks, and upon the last day with the intravenous give the intraspinous injection.

CASE III

The third case of cerebrospinal syphilis is interesting, as the two former ones were, not only from the standpoint of the results of treatment but also because it is complicated by a functional condition which is independent of the organic disease.

The combination of organic and functional disease is one all too commonly overlooked. In fact, I am inclined to think that if we look carefully for such symptoms we will find either functional exaggeration or independent functional manifestations in a great many diseases. This may be particularly true of diseases of the central nervous system; however, I am inclined to think it is true also of diseases elsewhere in the body.

The case history is as follows: A man thirty-seven years of age has been complaining for about a year of sharp, stabbing pains running down the anterior aspect of both lower extremities. He also complains of inco-ordination and general weakness.

The family history is of no importance. Sixteen years ago he had a chancre.

Physical examination shows that the pupils are irregular, the right larger than the left. They respond very slightly to light, and after the contraction the right pupil relaxes to its original size. They react well to convergence. The ocular movements are normal, also the facial, and those of the lower jaw. The teeth are negative, the tongue protrudes normally, the chest and abdomen are normal. The plantar reflexes are normal, the ankle- and knee-jerks absent. The superficial reflexes are normal, the wrist and biceps jerks present; triceps present, but diminished. There is no diminution of deep pain sense, that is, heavy pressure on the Achilles tendons, the testicles, peroneal, and ulnar nerves causes the normal amount of complaint. There is a superficial hypalgesia of the lower legs. There is no distinct diminution of the sense of passive movement.

Gait with the eyes open is normal; with the eyes closed he shows a curious tendency to fall toward the right, and is extremely apprehensive with regard to falling. It is this last feature that constitutes the functional complication.

Disturbances of gait are, of course, important symptoms in

organic disease of the central nervous system, as, for instance, the peculiar gait of the tabetic, the drunken gait of the cerebellar case. The gait in this particular case certainly is not that of the tabetic. There is practically none of the flopping of the feet as they are thrown forward, none of the stamping, the hyperextension of the knee-joint, or of the swaying when the eyes are closed. It resembles more the disturbance seen in the cerebellar case, that is, the falling to one side, but the obvious concern on his face, the outstretched hands, the fluttering eyelids, show that he is an apprehensive person, that he is keenly conscious of the abnormality in his gait. It has not been my experience to see these features in the true cerebellar case.

The pupillary changes are to be described as having the Argyll Robertson reaction, the loss of ankle- and knee-jerks, the history of chancre sixteen years ago, and the shooting pains, make the diagnosis of cerebrospinal syphilis of the tabetic type.

The disturbance of gait was considered as a tabetic manifestation by a doctor who had examined him previously, and before the patient I explained the difference between the true tabetic gait and the gait in this man. He was put on intravenous neosalvarsan and the trouble with his walking disappeared almost at once. Such rapid disappearance could not have been the result of the arsenic medication, and merely corroborates the diagnosis of functional disturbance of gait.

Several years ago I saw a patient who had the typical signs and symptoms of paretic dementia. His complaint, however, was that, though he could walk around his own room, the minute he got out into the hall he had to have somebody with him. It was not necessary that his companion should hold his arm, merely to touch the companion's hand was sufficient. He walked along the street with the outer border of his palm touching his companion. That sufficed. Obviously, this was a functional condition: if he could walk about his own room he could walk anywhere. I gave him some sodium bromid and told him I wanted him to take a walk alone the next day. He telephoned me at noon saying that it was wonderful medicine I had given him—that he had been out for a five-mile walk in the park.

Of course this had nothing to do with the organic disease except as a paretic is frequently as suggestible as an hysteric.

In this case before us there is unquestionably a high degree of suggestibility. The way the patient looks at the examiner with a combination of desperation and beseechment makes it obvious that he is willing to take any suggestion. This accounts for the improvement.

TWO CASES OF ENCEPHALITIS

I USE the term "encephalitis" without the modifying term "lethargica," because many of these cases show no lethargy. I would be inclined to modify the term with the word "epidemic," since there seems to be an epidemic of encephalitis at the present time.

Like all diseases of the brain, the symptomatology depends upon the location of the disease, and so in this epidemic of encephalitis we see cases of quite different types. Those first seen, at least so far as my experience is concerned, were all characterized by varying degrees of involvement of the extrinsic and intrinsic muscles of the eye: their pupils, associated movements, and regularity of movement—this last in contrast to the peculiar nystagmoid modifications of movement so characteristic of the condition. These cases doubtless depend for their characteristic picture upon inflammatory changes interfering with the regular and co-ordinated functioning of the oculomotor centers and the neural pathways, especially the posterior longitudinal bundle, leading between them.

I want to describe 2 cases of encephalitis which belong to the epidemic, but are symptomatically different from the more common type of the disease, in that the picture is dominated by disturbances of movement, especially involuntary, of the facial, trunk, and extremity musculature. I will describe the cases first and comment upon them later.

The first case was seen at Wesley Hospital on November 30, 1919. The patient was a boy eleven years old who had been complaining of pain in the right side of the face and neck, who had fever, thirst, vomiting, and convulsions. The history of the onset and course up to the time of entrance, as gained by the intern, was as follows:

Ten days before entrance the boy began to complain of a

sticking or pricking sensation like needles and pins in the right side of the face. Two or three days later he began to vomit. In the meantime he had been taken to a doctor who had prescribed some medicine; as to what the relation was between the medicine, the vomiting, and the thirst could not be determined. At any rate, there had been vomiting and thirst for several days before entrance. On the day of entrance fever was first determined; also, at about 5.00 A. M. on the day of entrance, the boy had a convulsion, these convulsions recurring during the day.

When first seen at the hospital the boy was rational at times, when he would complain of pain in the right side of the face; at other times he became quite delirious, shrieking and yelling at the top of his voice. When I first saw him he was lying in a position highly characteristic of meningeal irritation. The head was drawn back, the boy lying on his side, the knees and hips sharply flexed. There was no great rigidity or opposition to any passive movements, thus belying the posture in the sense that the latter indicated a meningitis. The pupils were dilated, reacting but very little, if any, to light. The reflexes could not be obtained because of the incessant movements of the patient when any part of his body was touched. He gave the appearance of being rather toxic, but it was evident that there was no distinct paralysis. There were no abnormalities of ocular movements; it was impossible to test sensation. The spinal fluid was under increased pressure and showed about 1500 cells to the cubic millimeter.

During the following six weeks the patient continued in a more or less irrational condition; he had frequent involuntaries of both urine and feces; there were no distinct convulsions. About two weeks after his entrance it was observed that he had not urinated for several hours, and examination showed a full bladder, which had to be catheterized. During this period of the stay in the hospital the white blood count varied between 22,000 and 16,000, the temperature between 104° and 100° F., the pulse between 150 and 110. The urine was negative. On January 12th he appeared much brighter; notes made at that time are

as follows: There is more or less continuous trismus, a slight spasm in the temporals and masseters, occurring about two or three to the five-second period, showing no distinct regularity. There are also frequent clonic spasms of the thoracic and abdominal muscles and the diaphragm. These spasms also seem to overflow into the musculature of the extremities, where, however, they are not nearly so prominent. The voice—and this is a matter of great clinical interest—is exactly like that of a patient with well-developed paresis; it has the same monotonous, slightly nasal quality, is tremulous, in the regular paretic tempo, and the syllables are not cleanly cut, mistakes in enunciation occurring in complex words. All voluntary movements are possible; the plantar reflexes are much diminished, but flexor in type. The right ankle-jerk is normal, the left is absent. The right knee-jerk is normal, the left diminished. It is impossible to demonstrate the abdominals and cremasterics because of the constant involuntary twitchings. He complains that a pin-prick feels like cold water on both lower legs. It is not felt at all on the anterior surface of the thighs. There is no Kernig, there is slight rigidity of the neck which seems almost a matter of volition: he will hold his neck rigid, then suddenly give up and allow the chin to flex practically to the chest. The mental confusion has entirely disappeared and he says that he can control his bowels and urine. Lumbar puncture at this time revealed no increase in pressure; the Ross-Jones was positive; 20 cells to the cubic millimeter.

On February 19th he said he felt quite well. He had been up and about for a week. Notes made at this time were as follows: The patient does not feel any involuntary movements. Upon examination, however, a peculiar complex, one is tempted to say co-ordinated, spasm or sudden contraction of the pectorals, the sternocleidomastoids, and the diaphragm is seen to occur about forty to fifty times a minute. At first it appears to be of an inspiratory nature, that is, as if it occurred with and was the cause of inspiration. When more closely observed, however, it is seen to be entirely irregular in tempo and not associated with the respiratory cycle except as the movements cause a partial

inspiration and expiration. He is able, for example, to take five deep inspirations without the spasms interfering with the rhythm; they do, however, interfere tremendously with ordinary quiet breathing. Associated with the movements of the above-mentioned muscles there are spasms of the latissimus dorsi on both sides, the left teres group, both biceps, the left much more than the right, and the left pronator and flexor groups of the hand. At times the adductors of the left leg twitch with the above-mentioned muscles; also there are irregular spasmodic twitchings of the rectus abdominalis on either side, at times together, at other times separately. There is also a convulsive twitch elevating the right eyebrow and retracting the right anguli oris. This is irregular in tempo, is markedly different in intensity at different moments, and occurs about eighty times to the minute. At times the movement around the eye is more marked, at others the movement around the mouth. There is a tremor of the tongue, both of the trombone type and a finer tremor along the edges of the tongue. There is no disturbance of sensation demonstrable; the plantar reflexes are normal, the ankle- and knee-jerks present, the right greater than the left. The abdominals are diminished, the deep reflexes in the arm are present on the right side, questionable on the left. The pupils and ocular movements are normal.

The description of the second case is as follows: The patient is a woman twenty years of age. There is nothing of note in the family history except that the father died five years ago of pleurisy. In the past history of the patient herself there was an attack of pneumonia at fifteen months; some dropsical condition at three years, cause unknown; whooping-cough and measles at five. None of these infections left sequelæ. She has been working as a stenographer for five years.

The onset of the disease was five weeks ago, about January 16th, when she gradually developed a stiff neck and a pain which ran from the left side of her neck down into the shoulders, to the heart region, and in the course of a few days extended on down the left side into the foot. This sensation was described as a sharp pain. As it was developing she was working very hard,

attempting to complete a piece of work in the office. As this pain began there developed a tremor of the hands and arms which gradually increased in intensity until, after a few days, it entirely prevented her feeding herself or even raising a glass to her lips. During this time there was no headache and no rigors, chilliness, or flushes to indicate a developing fever. Shortly after the onset she began to have what were described as "frightened spells," with screaming. These spells have occurred with considerable frequency, more often at night, and apparently more often when, for any reason, she was aroused emotionally.

There had been no diplopia or other difficulty with vision; no disturbance of speech had been noted.

Micturition had been difficult to start for three days previous to examination; also, careful inquiry into the past history of the case revealed the fact that frequently she would express the desire to urinate and when put on the pan would be unable to start the flow. This might happen a number of times before evacuation would be successful.

Examination.—The patient was first seen on February 23d, at which time the following notes were made: The patient is a thin, sallow complected girl, with a peculiar rash over the shoulders, chest, and buttocks. She is in a profuse perspiration and obviously much disturbed by the trip she has just made to the hospital. The rash is of a petechial appearance, and yet on areas of the skin where there are hairs it seems to be a fine folliculitis. This impression is borne out by the fact that her skin shows evidence of great neglect.

There is frequent, fairly rhythmic twitching of the adductors of the arms, each twitch causes an inspiratory movement which seems due to upward movement of the ribs. With this movement the recti abdominalis are stretched upward; the umbilicus moves upward about an inch; there is no apparent movement of the diaphragm. Synchronous with this spasmodic twitch there is a twitch of the muscles of the left leg, especially the quadriceps femoris and the extensors of the toes. There is also a slight movement of the flexors of the forearm and the pronators that occurs simultaneously with the twitch of the shoulder-girdle.

Voluntary movements of the eyes and the face are normal; the pupils are normal, the optic disk on the right side is redder than the left. Though I said that the movements of the face were normal, it seems as though the left face moved a little bit more than the right; the right appears a little washed out. This is true at rest, upon voluntary motion, and upon emotional expression. There is no resistance to passive movements, though there is slight rigidity of the neck.

The biceps jerks are normal, the triceps and wrist-jerks are absent, the right abdominal reflex is normal, the left absent. The knee-jerks and ankle-jerks are questionable; it would seem as though those in the left leg were absent and those in the right much diminished. The plantar reflex on the right side is normal, on the left is either stationary or slightly in extension.

Tests for sensation are interesting. She states that the pin does not feel nearly as sharp on the left side, especially the left leg, as on the right. The cold of an ethyl chlorid spray is felt as distinctly colder on the right side than on the left. It is difficult to demonstrate any change in heat sensation. Joint and tendon sense is apparently normal. The visual fields are grossly normal.

Along with the major or chief twitch or spasm previously described that involved the respiratory muscles and, to a less extent, those of the arm and leg, are constant, small tremor-like movements that seem to be associated with the chief twitch. This tremor-like condition is present upon active movements which, however, diminish the intensity of the chief twitch, For example, during ordinary quiet respiration the twitch, occurring as it does about fifty times to the minute, seems to be the only respiratory act. However, when forced and deep respiration is attempted the flow of air is but little, if any, interrupted, and the muscular twitch, as, for example, that of the pectorals, is distinctly diminished. The consequence is that an almost steady stream of air flows in and out in the course of a deep respiratory act, whereas with ordinary quiet breathing there is a very superficial respiration determined by the spasmodic twitch of the respiratory muscles.

The patient is well oriented and her memory is good. As

soon as allowed, however, she relapses into a more or less completely delirious condition in which she babbles unintelligibly and makes purposeless movements with the hands.

During the examination she had what has been spoken of as a "frightened spell." A look of terror and suffering came on her face, she cried out "Oh, my leg, my leg, it's so cold." There were no characteristic changes in the eyes, the body began a rapid clonic twitching, the left leg was partly flexed and held more or less rigid; there was a tendency to assume a position similar to that of the opisthotonos of hysteria. She reached for her mother, the mother being out of reach, then for the examiner. The respiration was rapid and a profuse perspiration broke out which soaked the sheet. The entire spell lasted about forty-five seconds. There was no loss of consciousness.

White counts on the 24th and 25th were 14,700 and 17,000 respectively. The temperature has ranged between 99.6° and 100.4° F. per rectum.

The lumbar fluid did not seem to be under a marked increase in pressure, there was considerable increase in the serum-albumin content, the Ross-Jones was positive for globulin. It could not be said that the cells were increased.

Comment.—These 2 cases seem to me to represent a condition of encephalitis where the inflammatory disease is localized in the basilar ganglia of the brain, possibly in the neighborhood of the red nucleus.

The involuntary movements in both cases strongly suggest those of respiration. The movements of respiration go on without the participation of consciousness. They are movements, one might say, of a lower order than those of speech or those of the finer functions of the hands and arms. They are, perhaps, to be thought of as even more automatic than the movements of walking. Respiration certainly continues during sleep; in fact, it is noticeable that people breathe much more deeply in their sleep than when awake, much more deeply automatically than consciously. Movements of this automatic sort probably have a center for their initiation far below the voluntary Rolandic cortex. It is highly suggestive that in this disease in which there

is an inflammatory reaction, especially at the base of the brain, that the spasmodic character of the movements is of this respiratory variety.

In both cases there are both objective and subjective abnormality of sensation to be demonstrated. In the first case a pin prick was felt as cold on the lower legs; in the second, pain and cold were diminished on the left side. I am inclined to believe that this sensory disturbance is due to an inflammation in the region of the optic thalamus. This inflammation might also account for the complaint in the first case of the pain in the right side of the face and in the second of the pain down the left side. I have not been able to demonstrate any change in the sense of passive movement nor in touch sense in either case. Disturbances of pain and temperature senses are especially characteristic of lesions in the thalamic region.

The attacks described as "frightened spells" in the second case may be hysteric; a combination of organic and hysteric symptoms is certainly much more frequent than commonly recognized. On the other hand, it may be that the attack represented some sort of an emotional discharge from the thalamic region brought on by an overwhelming emotional state; such a state orginating in the cortex in the form of an idea is transmitted to the thalamus where ordinarily it would receive its emotional tone; in this case instead of receiving the emotional tone it sets up a thalamic discharge. I cannot quite understand the complaint of the sensation of cold in her left leg at the beginning of the attack unless it is on some such basis.

Micturition.—In most of the cases of encephalitis that I have seen interference with evacuation of the bladder has appeared at some stage of the disease; in several cases the bladder symptoms have appeared only after the disease had progressed for ten days to two weeks.

Bladder centers have been postulated at various levels in the brain stem. It is probable that some center for the co-ordination of the various elements in the act of evacuation does exist in the lower brain.

The *initiation* of co-ordinated movements is not a spinal

function, also such initiation must, in some instances, occur far below the cortex. The cortex initiates the finer individual movements, as of the fingers, but respiration, heart-beat, swallowing, adjustments in the maintenance of balance, and even walking are probably cared for in the more automatically acting basilar ganglia. The initiation of micturition is a cortical function, but the co-ordination of the various elements is probably arranged in the brain stem.

Diagnosis.—It is obvious that in both these cases there is an infection; the temperature and the white cell count prove the presence of an inflammatory reaction in the body.

The posture, with retracted head, flexed thighs and legs, and the abnormal state of the spinal fluid point toward the meninges as being involved in the inflammation. The delirium, the subjective and objective sensory symptoms, and the involuntary movements are evidences of abnormal conditions in the brain. The spinal fluid in the second case was examined so long after the onset that the meningeal phase of the case might have subsided. The amount of meningeal involvement seems to vary, in some cases showing the posture as in case No. 1, and the marked pleocytosis, in others showing practically no signs.

At any rate, in these two histories there is sufficient to justify the assumption of an inflammatory disease in the brain.

The involuntary movements demand the differentiation from chorea. In chorea the movements show none of the constant repetition shown by the spasms in these cases, also the involuntary movements are seldom bilateral; one half the face will twitch, an eye will wink, the opposite shoulder will shrug, there will be a sudden jerk of the chin toward one or the other shoulder, etc., no rhythm, no repetition, no regularity, and, above all, no bilateral symmetry. The movements in these cases seem marked in the following particulars: the facial twitch in Case I was of the entire musculature innervated by the seventh nerve, not a wink or a grimace separately; the main or chief twitch was of the sternocleidomastoids, the pectorals, teres groups, and though in both cases the muscles of one side moved more than those of the other, still the muscles of both sides jerked simultaneously.

There is another form of so-called involuntary movement that must be differentiated from those seen in these cases. The movements of habit spasm or tic mentale are complex, co-ordinated movements that are frequently repeated.

As far as the movements themselves are concerned, it is very difficult to differentiate them from those of tic. The characteristics of repetition, of co-ordination of several muscle groups, the performance of an act that might seem purposeful, are common to both the respiratory spasm of these cases and to tic mentale. The fact that voluntary deep breathing only partially controlled the spasm, that the twitching of the muscles went on during deep breathing, speaks strongly against tic. Also neither patient was able to consciously reproduce the movement complex. The patient with tic can always reproduce the movement consciously.

In these cases the movements beginning with the onset of the infection mark them as something different from tic. It is usually possible to obtain an account of the beginning of a habit spasm or tic that shows it was first a conscious purposeful movement: shrugging the shoulders to lessen a discomfort in the back of the neck, blinking the eyes to relieve a smarting sensation, sniffing to relieve an unpleasant drying in the nose. There is no comparable element in these cases.

Treatment.—The most the physician can do is to give the patient the best chance possible to get well. There is no cure. It is wrong to give medicines that may in any way interfere with the patient's cardiac power or his respiratory power. Hence the coal-tar products are contraindicated except as they may be necessary to quiet the patient. Wet packs will secure the same results in most cases, with much less drain on the patient's reserve.

A daily colonic flushing is usually to be recommended. It may cause an evacuation of the bladder. In case the bladder does not work well, catheterization may be necessary for a few days.

It may be well to give a few intravenous normal salt injections, though there is no scientific reason for the recommendation.

The advisability of lumbar puncture may be considered here. There is some reason to believe that the withdrawal of a considerable amount of spinal fluid may predispose the meninges to infection, may make of them a *locus minoris resistentiæ*, so that germs in the body would be encouraged to attack them. This is not certain, but one should proceed cautiously, withdrawing only 2 to 5 c.c. of spinal fluid, just enough for test purposes.

CLINIC OF DR. WALTER W. HAMBURGER

MICHAEL REESE HOSPITAL

THE DIFFERENTIAL DIAGNOSIS OF CARDIAC AND GASTRO-INTESTINAL LESIONS, WITH PARTICULAR REFERENCE TO PECTORAL AND EXTRAPECTORAL ANGINA

ANGINA PECTORIS WITH JAUNDICE SIMULATING GALL-STONE COLIC

March 8, 1920.

I DESIRE to present this morning 2 cases which bring out several of the points of difficulty in differentiating between cardiac and gastro-intestinal conditions. While superficially one might think that such differentiation is readily made, and it must be admitted at once that in the majority of instances this can be done, nevertheless every once in a while we are called upon to see a patient in whom the findings and symptoms are so obscure as to make the decision between heart and stomach quite difficult, if not entirely impossible.

The first patient to whom I wish to call your attention is a man sixty-two years of age, an attorney, to whose home I was called in an emergency one morning at 4 o'clock. Upon entering his room I found this big, heavy man sitting doubled up in bed, clutching his right lower chest or upper abdomen as if suffering great pain. His family was anxiously hovering around him, and in their excitement stated that Mr. B. had wakened suddenly about an hour before with severe pain in the right upper abdomen which gradually increased in intensity, and shortly after its inception was followed by some nausea and finally vomiting. He had never had a similar attack.

My examination was quite difficult because of the tension he was under, because of extreme pain, and his difficulty in relaxing sufficiently to allow me to palpate and percuss him. His pulse was equal in both wrists, slow and regular, 68 to the minute. There were beads of perspiration standing out on his forehead and neck, and his color was rather a dusky red. His neck was short and thick; his heart borders were within normal limits except a possible slight extension to the left. The second aortic sound was increased; systolic blood-pressure was 180, diastolic 110.

Examination of his abdomen showed it to be somewhat distended and tympanitic, with quite definite tenderness in the upper right quadrant over the region of the gall-bladder. His reflexes and other physical signs were negative.

The pupils reacted to light and accommodation; the temperature was normal.

I felt that the diagnosis rested between a severe attack of angina pectoris, with atypical pain radiation, and an attack of gall-stone colic. Because of the continuation of the extreme discomfort he was given several hypodermics of morphin as well as nitroglycerin and pearls of amyl nitrite, and toward morning the pain gradually receded.

A more complete history obtained later stated that he had consulted physicians in his home town on different occasions for increasing shortness of breath and some slight precordial discomfort, particularly on walking upstairs and against a cold wind, although he had never had an attack in any way approximating the severity of the present one. However, from his increased blood-pressure and the history of preceding cardiac discomfort, and largely because of the intensity of the pain, I made a probable diagnosis of cardiac angina, and so told his family, although I must say that I felt considerable doubt regarding the possibility of gall-bladder disease.

The following morning about noon the patient was still suffering pain, although of less degree. He had a temperature of 99.4° F. and now had slightly more pain in his cardiac region as well as in the gall-bladder area. Twenty-four hours later a faint

but distinct jaundice developed, confirmed by positive bile tests in the urine, the temperature rose to 100° to 100.2° F., and because of the jaundice and temperature the difficulty in distinguishing between an angina and gall-bladder or liver disease became even more acute.

During the course of the next few days the patient steadily improved; the jaundice became slightly more intense, and then disappeared, the temperature gradually became normal, and within a week from the time of the onset the patient's condition was essentially normal. On the tenth day he was allowed to sit up out of bed, and on the eleventh and twelfth days he took a few steps around the house, and although the disease process involved had been somewhat obscure, I was totally unprepared for the event which followed. Two weeks from the onset I was again called over to the home in an emergency at 7 o'clock in the morning and found Mr. B. dead on the bathroom floor. He had gotten up to go to the toilet, called once to his wife, but was dead before she could reach his side.

One, of course, must conclude that this patient suffered with a severe angina pectoris, with probably some obstruction to his coronary circulation, with a resulting sudden and unexpected exitus.

The point that is of particular interest in this case is the differential diagnosis between angina pectoris and gall-bladder disease, and particularly the association of *jaundice* with a true angina pectoris. In looking this matter up, as this was a new experience to me, I found several references in the literature to similar cases, and if you will permit me I will read you a short description of 3 cases of Osler's which he included in the Lumleian Lectures on angina pectoris before the Royal College of Physicians of London, and reprinted in the Lancet, March 12th and 26th and April 9, 1910. The third case, that of "Dr. J. C. T.," is strikingly similar to the case I am presenting, namely, the ten-day interval between the onset and death, the development of jaundice on the fourth or fifth day of the attack, and the immediate and unexpected death.

"And lastly, I have a group of 3 cases in which attacks of

typical angina pectoris were complicated with abdominal pains, like gall-stone colic and jaundice. The combination may be quite accidental, but abstracts of the cases are worth recording.

"Captain M., aged sixty, seen January 13, 1897, had had in 1890 attacks of angina pectoris of the most characteristic type. In 1893 he had pains in the region of the liver with jaundice; these recurred at intervals, and he was operated upon by Mr. Mayo Robson at Leeds, 1895, but no gall-stones were found. He consulted me for pain in the abdomen, crossing from one side of the costal arch to the other, like a band or constriction; at times it became very aggravated. Food made no difference, and there was no tenderness on pressure and no enlargement of the liver. While more or less constant, the abdominal pain comes on in spells, and sometimes makes him feel faint and sick at the stomach.

"Mr. A. W., seen April 4, 1903, aged fifty-nine, in the first attack of angina four years ago had pains in the abdomen of great severity, and he was jaundiced after the attack. It was supposed to be gall-stones. Four months ago he had a very bad night, with attacks of pain in the chest and much discomfort in the epigastrium; on the following day he was jaundiced. The curious thing was that he could not differentiate the pain in this epigastric attack from the furious seizures of definite angina, in many of which I saw him during the six weeks that he was in the hospital. He died in an attack of angina some months later.

"Dr. J. C. T., aged sixty-four. After a day or two of indigestion and irregular pains had on October 27th a severe attack of pain in the chest, with extension into the arm, evidently of great severity. On the 30th the pain was more abnormal, and he was very sensitive over the region of the gall-bladder; on November 1st and 2d the pain was abdominal, and tenderness marked over the gall-bladder, and he had become jaundiced. This relieved our minds, as we were afraid, from the character of the early attacks, that it was angina. By November 9th he was very much better, and the pains had almost disappeared, and at 6 o'clock, when I saw him, he seemed nearly well; but at 8 o'clock, while his son and the nurse were in the room, he gave

one or two short, quick sighing inspirations, his head dropped on his chest, and he died instantly."

Neusser, in his monograph on Angina Pectoris, likewise mentions the association of anginal pain with cholelithiasis in the following words: "Radiation in the liver area can confuse angina with cholelithiasis. I personally remember a case of a young woman with aortic insufficiency who suffered with anginal attacks, following which attacks of nausea and vomiting occurred, tenderness to pressure in the enlarged liver area, and a subicteric color. My diagnosis was a complicating cholelithiasis in a valvular heart case. At autopsy no gall-stones were found, but a definite narrowing of the coronary vessels."

The explanation of the *jaundice* in angina pectoris I find extremely difficult. I have described this case to several professional friends, among them a pathologist, but have as yet been unable to find any satisfactory explanation. Unfortunately, an autopsy was not possible, so the heart and liver findings could not be obtained. In considering this case in retrospect, while the jaundice, the temperature, the tenderness over the liver region, and the right-sided pectoral pain were confusing, the heightened blood-pressure, the large, corpulent, thick-necked man, and, in particular, the extreme agony of the attack itself, together with the preceding history of slight dyspnea and precordial discomfort on exertion, were probably sufficient to point the way to a correct diagnosis. It is very possible that had sufficient weight been given to his complaint several years before, especially the difficult breathing and pain on walking against a cold wind, and if the possibility had been borne in mind that these were minor attacks of angina pectoris, something might have been done to regulate his activities of work and play, to reduce his weight, to modify his diet, so that his expectancy of life might have been prolonged.

I think the lesson such a case teaches to all of us is that in a man over fifty with findings such as these too much consideration cannot be given to even the slightest discomforts, and if one constantly bears in mind the possibility of such cardiorenal lesions, proper interpretation and care will follow as a matter of course.

ANGINA ABDOMINALIS WITH CHRONIC DUODENAL ULCER SIMULATING CHRONIC GALL-BLADDER DISEASE

The second patient is equally interesting. A merchant, fifty-eight years of age, complained of attacks of pain in the upper abdomen since 1906. The pains came on from an hour and a half to three hours after eating, and were relieved frequently by sodium bicarbonate and by having small meals. He complained likewise of marked constipation, belching of gas, and gradual loss of weight. He weighed in 1911, 240 pounds; in 1916, 215 pounds; in January, 1919, 202 pounds; at present (1920), 164 pounds. There was no nausea or vomiting and no distress of any sort between his attacks of pain, which came on periodically, often following prolonged exertion, business trips, and strain and worry regarding business affairs. I first saw him about three years ago, at which time a diagnosis of colitis, chronic cholecystitis with adhesions about the hepatic flexure was made. Under bed rest, a strained diet, and belladonna he made a prompt and apparently complete recovery. He was examined again in the hospital in May, 1919, at which time the earlier diagnosis was confirmed, a more definite diagnosis of gall-bladder trouble was made, and an operation for draining the gall-bladder was advised. From May, 1919 until December, 1919 he continued to have periodic attacks of pain with loss of weight, and consulted a number of different internists and surgeons, all of whose opinions coincided, to the effect that he probably suffered with chronic gall-bladder trouble, necessitating operation. He was operated on December 22, 1919. The gall-bladder was found essentially normal. In the first portion of the duodenum there was a large, deep, callous ulcer without adhesions, and for which a gastro-enterostomy was performed. Exploration of the abdomen otherwise was negative. I should have stated earlier that *x*-ray examination of his stomach made on several different occasions had revealed no defect in the duodenum, no obstruction to the emptying time of the stomach, and his test-meals were normal. Chemical blood was never found in the stomach contents or stools, and the finding of the ulcer was a complete surprise to all who had been observing this case.

The patient made a very stormy postoperative recovery, vomiting, having extreme discomfort from gas-pains, and continuing to do poorly for several weeks. Even after he was able to take small amounts of food, his diet being changed from time to time in an attempt to obtain for him an acceptable diet, he would constantly have recurring upsets of rather sharp, colicky abdominal pain, for which various explanations were offered; namely, that his appendix was now troubling him, that his colon was again upset, that he was suffering with acute dilatation of the stomach, etc. We thought that he might have an atypical ulcer picture, and three weeks after operation he was put on small hourly feedings of milk and cream with alkali between feedings, with the result that for a short space of time he obtained relief.

A typical attack of pain occurred February 4th and may be described as follows: "8.30 P. M., three hours after his supper at 5.30 (one soft-boiled egg, strained rice, and apple sauce) there is a dull ache which radiates downward and sideways. Feels like gas-pain. At times continuous. Tendency to radiate to both shoulder-blades; is as severe as a labor pain. The patient, during the peak of the pain, has beads of perspiration on his forehead. The pulse is steady, regular, and slow. The patient is extremely apprehensive and insists on sending for his family. Systolic pressure 104, diastolic 65."

A recurrence of this pain with elevation of blood-pressure and prompt and complete relief with nitroglycerin led to the correct diagnosis, as follows:

"February 22d, during an attack of pain at 2.10 A. M., systolic pressure was 154, diastolic 102; patient extremely nervous and excited. At 2.20 A. M. $\frac{1}{50}$ gr. of nitroglycerin was given on the tongue. The pressure dropped, systolic 142, diastolic 80; patient more calm; 2.40 A. M. systolic pressure 123, diastolic 86; pain almost gone." The relief obtained with nitroglycerin confirmed the surmise that an angina abdominalis was responsible for the continuation of his discomfort, and the subsequent history proved the correctness of this view. Following this experience Mr. Y. was put on 10 grains of diuretin four times a day with $\frac{1}{50}$

gr. of nitroglycerin as necessary for acute attacks, with prompt and complete relief of all symptoms. From this point on he made an uneventful, steady recovery, the nitroglycerin being necessitated only very occasionally and the diuretin being constantly decreased.

At the present time the patient is on a full diet, has gained 12 pounds in weight, and is about ready to resume his business affairs. It seems quite certain that he suffered from a typical angina of the type that has been called angina abdominalis, and from which relief was prompt and satisfactory under diuretin and nitroglycerin.

Angina abdominalis is now pretty well recognized, particularly through the work of Ortner, Muller, and Neusser abroad and Gilbride and Akin in this country. The condition may be a true atherosclerosis of the abdominal vessels giving an "extrapectoral angina," as Osler calls it, or may be a true angina pectoris with abdominal localization. Akin describes the patients as presenting marked weakness and loss in weight, and Ortner comments on the fact that attacks of intense abdominal pain are often precipitated by food, particularly the cabbage, pea, and bean variety. A woman in Neusser's wards passing through such an attack reminded one of the agony of gastric crises or gall-stone colic. Sudden profuse hematemesis has been described. As a rule, the blood-pressure is increased, particularly during the height of the attack. I have found no reference to cases simulating gall-bladder disease.

This case is of special interest in view of the generally low blood-pressure readings, notwithstanding which relief was obtained with diuretin and nitroglycerin. In some experimental work on dogs several years ago I was able to show that intravenous injections of moderate amounts of diuretin caused a fall in the carotid blood-pressure, apparently from splanchnic vasodilatation, and suggested that this might explain the relief obtained clinically in angina abdominalis following the administration of the drug.

The most interesting angle to this case, it seems to me, is the fact that here was a patient, observed for several years, having

the benefit of the most modern clinical and laboratory methods of diagnosis, seen in consultation by several foremost internists and surgeons, going under a competent surgeon's knife for a gall-bladder operation, having a gastro-enterostomy performed for an ulcer, all without relief of the symptoms, and, finally, being relieved by very simple drug measures. All of us who have been interested in this patient feel that unquestionably this abdominal angina explains most if not all of his symptoms, and that if this had been recognized earlier the patient might have been spared long suffering and considerable surgery. A case of this sort again emphasizes the difficulties in diagnosis in obscure abdominal cases, perhaps, in particular, those of the upper right quadrant. The frequency of gastric crises in tabetics, masquerading as gastric ulcer, gall-bladder disease, and other abdominal pathology, is well known, and only rarely does one now see tabetics with abdomens scarred with surgical incisions. This case teaches the importance of considering abdominal angina with vascular crises giving abdominal pain as a possible frequent similar source of error. I personally feel that hereafter in all obscure chronic abdominal cases associated with periodic colicky pain, even if the evidence points pretty clearly to organic disease, that before allowing such a patient to submit to operation a therapeutic test of diuretin and nitroglycerin should be made.

I would like to offer this suggestion for general use, and would be interested in knowing the experience of others if such therapeutic tests were made.

Just one word more. These two cases of *apparent* abdominal disease, one a "cholelithiasis with jaundice," the other a "chronic cholecystitis" with ulcer, proved in the first case to be true angina pectoris, and in the second, angina abdominalis. They serve very well, I think, to bring up the subject of the differential diagnosis between cardiac and gastric conditions in certain cases, and I believe if one simply has in mind their mutual relation and interrelation, one will not be apt to go astray. Within the past year I should say I have seen, in addition to these two, perhaps four or six similar cases where, on the first impression, it was exceedingly difficult to decide whether one was dealing

with pathology above or below the diaphragm, although a careful history and examination usually resulted in the correct interpretation with corresponding therapeutic result. I may be permitted to recall one very striking instance of an old man with an enlarged, tender mass in the upper right quadrant of the abdomen, which the surgeon considered an inflammatory tumor of some sort, and advised operation, but which under bed rest and digitalis very promptly receded, and proved to be an engorged, tender liver from heart muscle failure. Instances of this kind are perhaps not infrequent and in the experience of all. All I have hoped to do today is to once again call your attention to the possible frequency of error in the differential diagnosis of these two conditions.

CLINIC OF DR. JOSEPH C. FRIEDMAN

MICHAEL REESE HOSPITAL

CALLOUS ULCER OF THE STOMACH

Full Discussion of Diagnosis; Causes of Gastric Pain; Management and Treatment.

THE first patient whom I wish to demonstrate today has returned to the hospital at my request for a few days in order that we might determine his present condition as accurately as possible. He originally entered the hospital April 29, 1906, on the service of Dr. M. L. Goodkind, being twenty-seven years of age at that time, with a history of epigastric pain which had recurred at intervals during one year. It had been particularly severe for the two weeks immediately preceding his entrance to the hospital, especially after meals, radiated to the back, was accompanied by nausea and at times by vomiting, especially in the morning. There was no blood in vomitus or stools.

Examination disclosed nothing of importance except a point of tenderness in the midepigastrium. An Ewald meal showed a total acidity of 73, free acidity 48; blood positive chemically. The diagnosis of gastric ulcer was made, the patient treated accordingly, and discharged May 20th. He apparently was very efficiently treated, for he had no gastric disturbance of any kind for over eight years.

It may be of interest to note that his diet during his first hospital stay consisted of peptonized milk and beef juice per rectum, and that no alkalies were administered by mouth.

He returned to this hospital in October, 1915, having then had pains for about one year, resulting eight months previously in an exploratory laparotomy elsewhere. No ulcer was found, but the pylorus seemed to be thickened, and a pyloroplasty was

done. He felt better for six months, when the distress returned and has persisted. The pains were about the same as those of nine years before, beginning two and a half hours after a full meal, relieved by soda, often awakened him at night, and caused a loss of weight of approximately 10 pounds, owing to the fear of food. Venereal infection was denied.

Examination again revealed the same tenderness in the epigastrium to the left of the laparotomy scar. Blood Wassermann test was negative. An Ewald meal showed a total acidity of 70, free 53. No blood was present. Motor meal, no residue in six and a half hours. Stool, blood present once in three examinations.

A test-meal consisting of 300 c.c. of tea and 10 gm. of lactose was given for fractional aspiration, the fractions being removed every half-hour for three hours, with the following results:

Time.	Free acid.	Total acid.	Bile.	Lactose.
8.00	56	80	0	+
8.30	40	62	0	+
9.00	25	40	0	+
9.30	0	12	+	0
10.00	0	14	+	0
10.30	0	10	+	0

The first two fractions are markedly above the normal, while motility is normal, the lactose persisting for one and a half hours. In the first *x*-ray picture (Fig. 259) there is a typical niche in the posterior wall close to the lesser curvature in the pars media. It will be remembered that the symptoms are typical of the pyloric and duodenal syndrome; that is, late pain, night pain, etc., and still the ulcer is quite near the cardia. Another illustration of the well-established fact that the time of the pain need bear no relation to the situation of the ulcer. We may have early pains with duodenal ulcer and late pains with ulcers near the cardia. The antrum close to the pyloric ring is defective, and the bulb does not fill well. Motility is normal.

The diagnosis then was callous ulcer of the lesser curvature. Such ulcers are, we know, the least amenable to medical treat-

ment and, moreover, such treatment had been carried out *lege artis* elsewhere just before his operation, with no result; so a second operation was advised. It was only after most persistent search that the operator, Dr. L. L. McArthur, was able to find the ulcer, well toward the cardia and adherent to the pancreas. It was resected and the wall of the stomach sutured. The patient left the hospital with still some gastric distress, and re-entered in December, 1915 for a few days.

Fig. 259.—Callous ulcer.

Another fractional meal at that time, consisting of tea and lactose, gave about the same figures:

Time.	Free acid.	Total acid.	Bile.	Lactose.
8.00	16	26	0	Trace
8.30	66	78	0	+
9.00	45	55	0	Slight trace
9.30	20	44	0	0
10.00	30	50	0	0
10.30	22	42	0	0

Namely, hyperacidity plus normal motility. Fluoroscopically he showed a narrowing at the point of resection and normal motility. The pylorus and bulb were defective, as before.

He re-entered the hospital in March, 1916, with the history of almost *continuous distress* in the right side and back, diffuse, stabbing pains, and occasional vomiting. In short, the same symptoms as before the operation, but more intense. Fasting aspiration at this time yielded 100 c.c. gastric juice, free acidity 54, total 102; 75 c.c. N/10 HCl given through a tube relieved the pain about as well as bicarbonate of soda, though the next day after the administration of the acid was a particularly bad one.

A tea-lactose fractional meal gave a maximum total acidity of 52 and 50 in the second and third fractions. The bismuth meal at this time showed a partial hour-glass stomach, and on the lesser curvature a niche with the incisura on the greater curvature. Evidently a new ulcer had formed at the site of the one excised.

He was operated again in March, 1916 by Dr. E. Wyllis Andrews. Massive adhesions were found between the stomach and the anterior abdominal wall, about the lesser curvature and the pylorus. The posterior wall was greatly thickened, and the crater of the ulcer could be palpated. A gastro-enterostomy was done with considerable difficulty. He was discharged in May, 1916 in good condition, but still with pain in the abdomen occasionally in the evenings. The absence of complete relief after this operation was considered to be due not only to the presence of the ulcer but also to the massive adhesions which practically surrounded the stomach. He returned to the hospital in September, 1916, stating that for the past week the pains had been very severe, especially in the right hypochondrium, epigastrium, and back.

A fractional meal at this time gave the following figures:

Time.	Free acidity.	Total acidity.	Sugar.	Bile.
8.00	0	10	+	+
8.30	0	14	+	+
9.00	13	28	+	+
9.30	13	29	0	+
10.00	27	37	0	+
10.30	18	23	0	+
11.00	21	27	0	+

The usual low acidity following gastro-enterostomy. This fact, coupled with the severe pains, should be borne in mind in view of the present findings to be considered later. The *x*-ray (Fig. 260) showed the following: The gastro-enterostomy opening is patent. The niche is still present on the lesser curvature. Several incisuras are noted in the region of the antrum. The bulb is not well seen and the bismuth passes readily through the duodenum.

Fig. 260.—Callous ulcer.

Three weeks later he was placed on a gastro-intestinal diet, which consists of food in finely divided form, all vegetables being passed through a hair sieve, and the meat through a fine chopper. The pain recurred. Again, he was placed on a milk-cream mixture, without result, and on October 21, 1916 an Einhorn tube was passed into the jejunum and the patient was fed entirely through this. His weight at this time was $121\frac{1}{2}$ pounds. He was given milk and cream, broth, gruel and eggs, and fed every two hours. On the 25th his weight was $116\frac{3}{4}$ pounds; 2 per cent. lactose was added to the milk, cream soups

were given, but his weight continued to drop, reaching its minimum, 114 pounds, on the 27th. His weight then began to increase, and on the 29th was $116\frac{1}{2}$ pounds. At that time he was getting eight feedings daily, 6 ounces to a feeding, consisting of milk three-fourths, cream one-fourth, plus 2 per cent. lactose, 3 eggs, farina and cream soup, a total of about 2000 calories. On November 9th the tube was finally removed, after almost three weeks' feeding, his weight then being $126\frac{3}{4}$ pounds. He was placed on a finely divided solid diet and discharged.

He returned January 18, 1917, saying that the pains had returned a few days after leaving the hospital. The same tenderness was present in the right hypochondrium. His weight on entrance was 128 pounds. He was fed as before, through the duodenal tube, about the same diet with some beef juice, and on February 8th his weight was $131\frac{1}{4}$ pounds. The pains had ceased and he was discharged on the 12th. He was given a duodenal tube which he passed morning and night, eating nothing during the day while at work. Owing to the gastro-enterostomy opening there was no difficulty in passing the tube directly into the jejunum, a point which was verified several times fluoroscopically before his final discharge. He kept this up for several weeks after leaving the hospital, losing some weight, but keeping comparatively free from pain. As you see, his color is good. He assures us that he rarely has a pain, though now he is quite liberal with his diet. The *x*-ray (Fig. 261) now shows no niche. The antrum is barely outlined and the stomach empties chiefly through the gastro-enterostomy opening. It is absolutely immobile, however, owing to the dense adhesions. The tea-lactose test-meal gives the following figures:

Time.	Free acid.	Total acid.	Sugar.	Bile.
Fasting Aspirator....	0	13	0	+
11.15.....	18	38	+	+
11.45.....	38	59	+	+
12.15.....	32	46	0	+
12.45.....	21	38	0	+

A rather high acidity then for a stomach with a gastro-enterostomy; higher, in fact, than in 1916, when his pain was at its

height. Then, you will remember, the maximum for free acid was 37 in the fifth fraction. At that time the outlook was considered most discouraging, it being argued that even were the ulcer to heal the pains would continue probably unabated owing to the dense adhesions.

Such was not the case. The ulcer has apparently healed, as the niche is no longer visible, as you can see in the plate (Fig. 261).

Fig. 261.—Callous ulcer.

and the patient is almost entirely free from pain in spite of the fact that the adhesions still remain, and there is immobility of the stomach fluoroscopically. The question as to the cause of pain in gastric ulcer is still unsettled. The theory that it is due to a hyperacid gastric juice acting on the exposed nerves in an ulcer base has many objections, but has never been completely refuted. The objections are obvious enough:

1. We can get typical ulcer pains after ulcers have healed, leaving only scars. This is more often seen perhaps in duodenal ulcer. The adherents of the theory insist that the scar may be as sensitive to acid as the original ulcer.

2. We may get typical ulcer pains in achylia gastrica with, of course, no free HCl in the gastric juice. This was long ago pointed out by Einhorn.

3. It has repeatedly been shown, and recently very clearly by Krohn, that clinical improvement may take place with complete disappearance of pain, yet hypersecretion and hyperacidity persist unaltered. The adherents of the theory answer that it is not hyperacidity *per se*, but a hyperacidity acting on the ulcer base which causes the pain, and that in the cases cited the base became at least covered with epithelium, and desensitized as it were, so that no pain was caused even though the hyperacidity remained.

4. It is often argued that because the pain is so often relieved by alkalies that, therefore, it must be due to the acid factor in the gastric juice. That is not the whole truth, however, for, as mentioned before in this case, the pain could also be relieved by pouring dilute HCl through a tube into the stomach.

It was to meet such objections that the theory of gastric pain due to increased peristalsis with increased intragastric pressure was advocated by Lennander, Hertz, Carlson, Tumpowsky, Hamburger, and others. There is, of course, no question that there are violent peristaltic waves often present in the stomach at the time the patient complains of pain. More recently Homans has shown that ulcer pains may be very pronounced when no active peristalsis is present. As he points out, however, the method employed takes account only of contractions in the fundus, while dynamically the pyloric end of the stomach is the most essential factor. In our patient a comparison of the second and third plate (Figs. 260, 261) shows that at the period of greatest suffering, when the second plate was taken, the pyloric end of the stomach was well filled, and that at present when free from pain there is barely enough barium in the distal end of the stomach to outline it, practically all of the meal passing

through the gastro-enterostomy opening at present. It may be that this inactivity of the antrum has something to do with the diminution in pain. I do not consider the case as proving either theory. However, another cause of pain must be considered, and that is perigastritis or gastric adhesions. Such pains are not due to irritation of the sympathetic, which is probably the afferent nerve in intragastric pain, but to the posterior roots of the spinal nerves supplying the subperitoneal tissues. Adhesion pains can often be identified by the following characteristics: they are more continuous than contraction pains; they occur earlier—in fifteen or twenty minutes—after eating, and are less likely to be relieved by taking food. These points are easily explained if we remember that they are probably due to pressure or pull on the subperitoneal tissues. However, the importance of adhesions in producing pain anywhere in the abdomen is probably greatly overestimated. The phenomenon observed here is one which I think most surgeons are familiar with; that is, that when the active inflammatory process causing the adhesions is removed, in this case the healing of the ulcer, the pain ceases, the presence of scars fixing normally mobile organs not being ordinarily a cause of pain. The same statement applies to other viscera, as the gall-bladder and appendix. The viscera adopt themselves to the new conditions most remarkably, except, of course, where actual stenosis is produced, when the pain would be due to hyperperistalsis. When no stenosis is present the adhesions simply result in hypersensitiveness of the viscus. Pains will arise on slighter provocation than in normal individuals, and they are obliged on that account to be more careful in the selection of their diet.

The conclusion, then, is plain. In perigastritis due to an active callous ulcer the ulcer should be excised when possible and a gastro-enterostomy done. In perigastritis due to a healed ulcer gastro-enterostomy alone is sufficient; freeing old adhesions is superfluous.

Another point of interest is the effect of pyloroplasty on acidity. Mikulicz, over twenty years ago, claimed that in a number of ulcers with hyperacidity a simple pyloroplasty caused

the acidity to gradually drop to normal. Such was not the case here; the acidity did not diminish until the gastro-enterostomy was done. Mikulicz's statement is not to be doubted, however. It is very possible that the acidity in the cases mentioned by him diminished because the ulcer healed after the pyloroplasty was done, and the hyperacidity then disappeared. In other words, hyperacidity is very probably in many cases a result and not a cause of ulcer. Treat the ulcer and the hyperacidity will take care of itself.

This brings us to the question of ulcer treatment. The points brought out are these: Fourteen years ago rectal feeding, that is, complete gastric rest, for two weeks brought about a remission of over nine years. Nine and ten years later strict ulcer diet with milk and cream mixtures and alkalies had no effect. Again, complete gastric rest by duodenal feeding, and no alkalies, and again apparent healing.

First of all, the value of alkalies in the treatment of ulcer is probably overestimated. They do not promote healing directly by neutralizing acidity. They do so by diminishing peristalsis, relaxing the pylorus, and so placing the ulcer at rest. The work in the Carlson Laboratory at the University of Chicago is most important in this connection. Here experimental ulcers were produced in the lesser sac of a Pawlow stomach. The pouch was then fixed so that the ulcer remained constantly submerged in undiluted gastric juice. In spite of this the ulcer healed rapidly and completely. Another type of evidence can be deduced from clinical observation. How often do we see ambulatory cases treated with milk and cream and alkalies *ad libitum* with no result, and then by adding rest in bed, perhaps with compresses, produce immediate improvement? In other words, we have a chronic inflammatory process the healing of which is inhibited by mechanical rather than chemical irritation. I would not on that account discontinue the use of milk and alkalies. It is a very simple and effective method for use in the majority of cases of ulcer. I wish, however, to emphasize the fact that while effective, there is little well-founded evidence in favor of the commonly accepted theory of its method of action. Giving

alkalies is simply one method of reducing spasm which interferes with the blood-supply of the ulcer area, and so prevents regeneration of the destroyed tissue.

We must, therefore, bear in mind the fundamental aim in keeping the stomach at rest, and where the simplest measure fails, resort to more stringent ones. The first method used was, of course, rectal feeding. The clinical objection to this for any prolonged period is that not more than 600 or 700 calories can be absorbed daily in this way, and that it is, therefore, a starvation method, and that possibly healing may be interfered with in such devitalized tissues, and that unquestionably convalescence is prolonged. No such objection can be raised to duodenal feeding, as sufficient calories may be easily introduced and absorbed by this method. It is not practicable as a routine method, however, as many patients prefer the ulcer to the tube, and should be reserved for the more refractory cases.

Even in the simple cases, and these constitute the majority, I am not an advocate of prolonged milk cures. In such cases it is desirable in five to seven days to begin eggs, gelatin, and custard, and in seven to ten days finely divided cereal, powdered vegetables, and very minutely divided meat of the short-fibered kinds. Patients can thus be taught the dietetics of their disease before leaving the hospital, and are more apt to observe the rules later on. Where these methods fail, or where dietetic rules cannot later be observed, surgery should be resorted to.

There is one more point which cannot be brought to mind too frequently, and that is the unreliability of statistics, medical or surgical, of cures in ulcer cases. In a disease subject to both spontaneous and induced remissions, often for many years, statistics covering only a few years can have little value.

CHRONIC NON-SPECIFIC ENTEROCOLITIS

Types of the Disease, Etiology, Full Discussion of Treatment.

THIS patient is in a very different condition from the previous one. We hope that she is on the road to recovery, but evidently the goal is still distant.

She is forty years of age and single. Her illness began five years ago with pain in the left lower abdomen, nausea, and diarrhea. The pain was of a sickening, griping type, and more prominent during the frequent bowel movements. There was nothing grossly abnormal in the stools; that is, no mucus or blood. Whether there was on more careful examination the patient does not know. The condition was diagnosed colitis, and she was placed on a diet avoiding bulky foods. After three years of varying improvement and relapses the diarrhea became very severe, continuous bowel movements throughout the forenoon, associated with severe, griping pains, which subsided in the afternoon and evening, when the bowel movements diminished in number. The stools during the third year contained much mucus, but no gross blood. At this time, 1917, a tumor developed in the lower left quadrant, with severe local pain, difficulty in walking, and almost complete inability to bend forward. She was operated, and a large ovarian cyst removed, together with the appendix. For a month after the operation the condition seemed improved. There were three formed stools daily, with grossly visible mucus only with the first. After a month the stools began to show blood, visible in streaks and larger masses of mucus. The diarrhea increased in intensity. During the summer of 1918 the patient had subcutaneous injections of streptococcus vaccine and apparently improved for a while, but during the winter mucus and blood were present in varying amounts. In June, 1919 she says she became severely ill, with a temperature of 102° F., intense headache, pallor, severe abdominal cramps, but only two or three bowel movements daily.

In September a severe diarrhea developed, twelve to fourteen bowel movements daily. She was taken to a hospital for observation and *x*-ray examination. The latter showed nothing of importance, apparently. The patient was placed on a gruel diet and in nine days lost 8 pounds, going from 112 to 104 pounds. She went home and was kept on daily quinin enemas and given everything she could eat. Her bowel movements averaged fifteen to eighteen daily; pain was severe in both right and left flanks, and flatulence was very distressing. This continued until November, when nausea set in and severe headaches. At that time she entered the hospital.

She was extremely emaciated; the skin was dry and dark, without areas of more intense pigmentation. The mucous membranes were normal; no pigmentation. The lungs and heart were normal, the pulse ranging from 70 to 80.

The abdomen was somewhat distended and tympanitic. The outlines of the transverse and descending colon were indistinctly visible, but without abnormal peristalsis. There was some tenderness in the left hypochondrium and right iliac fossa. There was no rigidity and no tumor was palpable. The liver and spleen were not palpable; the right kidney could be palpated.

Proctoscopic examination showed an intensely hyperemic ampulla, with thickened, easily abraded mucosa, but no ulcers or scars.

Gastro-intestinal *x*-ray examination done previously had resulted in no organic abnormalities. It has not been repeated, owing to the patient's precarious condition.

The laboratory results were as follows: The urine showed a trace of albumin and a moderate number of pus corpuscles. Triple agglutination test on blood negative. Blood Wassermann negative. Hemoglobin 70 per cent., red blood corpuscles 3,700,000; white blood cells 10,600, with 63 per cent. polymorphonuclears. Blood-cultures were negative. The stomach contents show a free acidity of 10, total 24. At this time the temperature was intermittent, from 99.4° to 103.8° F. daily, the pulse ranging from 96 to 120. The blood-pressure was 108 systolic, 70 diastolic.

The stools at this time varied from 4 to 8 ounces each in amount and from thirteen to sixteen in number. They had a yellowish-green or yellowish-brown color, evidently from unreduced bile, and were rather sour in odor. They were slightly alkaline or slightly acid in reaction, with marked increase in mucus, partly intimately mixed with the stool, giving it a thick, stringy consistency, and partly in separate, finely divided particles. Food remnants were not in evidence. Very numerous soft, white particles floated in the liquid, which proved to be masses of pus corpuscles with varying numbers of red cells. From time to time quite large, dirty white masses of soft consistency resembling sloughs of mucosa appeared in the stool. These histologically proved to be simply masses of pus-cells embedded in fibrin with many red cells and some lymphocytes, but no organized tissue elements. No tubercle bacilli and no amebæ found on repeated examinations. Only Bacillus coli could be grown in cultures.

In short, we had a very violent inflammatory process of the intestinal tract, in which large amounts of pus were constantly discharged, yet it was not a localized abscess, as was shown by exploration during the laparotomy, but a diffuse intestinal condition, in which no specific invader could be determined.

As to etiology, the evidence here is mostly negative. Having eliminated the specific ulcerative conditions by both the laparotomy and the examination of the stools, a diarrhea from an achylia gastrica by examination of the stomach contents, as well as by the fact that the latter never produces such violent hemorrhagic conditions, we must consider the possibility of a ductless gland disturbance, here more particularly an Addison's disease. The patient was intensely prostrated. She was not able to lift her head from the pillow without becoming dizzy, and certainly there was a marked, diffuse pigmentation of the skin. However, this pigmentation was not more pronounced in the genitalia, waist-line, etc., and was not present at all on the mucous membranes of the mouth. The systolic blood-pressure was over 100, and here again the violently inflammatory nature of the process speaks against such a presumption. We were forced, then, to fall

back on a non-specific intestinal infection, with a diffuse general involvement of the mucosa and underlying tissues. We conclude that the mucosa must be quite deeply involved, owing to the long-continued, violent inflammatory reaction, as evidenced by the stools. In determining the localization in the tract we make the classification into: (*a*) small intestine catarrhs; (*b*) diffuse processes involving both small and large intestine, and (*c*) colitis. The characteristics of (*a*) are the marked subjective disturbance; that is, the meteorism, belching, and colicky pain, and, especially in involvement of the ileum and jejunum, the light brown, or yellowish-brown, foaming, acid stool with a sour odor, rather large and free in number, with evidence of undigested fat, protein, and starch. Mucus may or may not be present. Where the diarrhea is continuous, from three movements upward, and the stool presents some of the foregoing characteristics, it is an evidence of type (*b*); that is, a diffuse process. Here, however, tormina is apt to be present, and the stools become smaller and more numerous as the sigmoid and rectum become irritated. The mucus remains intimately mixed in the most frequent localization, *i. e.*, the combination of ileum, cecum, and ascending colon. Indeed, the frequent relapses in these cases are due, generally, to residual processes left in the cecum and ascending colon. Of course, the lower the inflammatory process, the greater the tendency of the mucus to be in larger masses and more separate from the fecal matter. In type (*c*), or the purely large intestinal type, while there may be alternating constipation and diarrhea, in the more frequent irritable form there is a stubborn diarrhea, similar to dysentery, with blood in the stool, and often with fever.

In continuing the history of this patient from her entrance into the hospital I shall point out the facts which indicate a change from type (*b*) to type (*c*). Since her entrance her temperature has gradually subsided, with occasional remissions, however. For instance, on December 7th the maximum was 101.4° F., the minimum 98° F. On December 22d, 98.6° to 99.6° F. January 3d, maximum 99.4° F., minimum 98.6° F. Since February 14th the minimum has been 97.6° F., the maxi-

mum 99.2° F., until two weeks ago, since which time it has not been above 99° F., and generally normal. Coincidently with this has been a decrease in the number of stools. In the early part of December they averaged twelve to fifteen daily, with the qualities already mentioned, namely, rather large, dark green, with mucus and masses of pus. In January they averaged ten or eleven, and toward the end of February seven or eight, with, of course, short periods of exacerbations. In January, for instance, for several days there were fifteen and sixteen stools daily. Concomitantly there has been a change in the size. In November the early morning stools would average 7 and 8 ounces. In January at one period the maximum was 4 ounces, the others being 2 and $1\frac{1}{2}$ ounces in size. About this time also the headaches and nausea and general abdominal cramps disappeared, and foaming and tormina and pain in the rectum appeared, the stools became foul in odor, alkaline in reaction, and the mucus increased enormously in amount and floated in large masses on the surface. This is the change before spoken of, from type (*b*) to type (*c*), characterized by diminution in subjective symptoms, by disappearance of unchanged bile—that is, change from dark green to brown or yellow, by change in reaction from acid to alkaline, by increase in visible mucus, and, furthermore, the involvement of the pelvic colon and the rectum was indicated by tormina, small stool, and rectal pain. At present the stools average about seven daily, the maximum is about 5 ounces, light brown, slightly acid, or slightly alkaline, sour odor, but a great diminution of microscopic pus and blood-corpuscles. A few yeast cells and molds are also seen.

The history of the case is, I believe, to be interpreted as follows: Five years ago the patient developed a simple colitis, probably an infectious catarrhal process, on the basis of a disturbance due to pressure of a developing ovarian cyst. Two years ago, at the time of rapid growth of the cyst, the infection extended, mucus became more pronounced, and pain more severe. Last fall the process became more acute and still more extensive. The small intestine became involved, either by direct extension or reflexly, for the first time, as indicated by the flatulence,

nausea, headache, etc., the color of the stool, and the intensity of the infection by the temperature, and the presence of pus and blood. As mentioned before, the small intestinal process subsided and the whole large intestine became involved, including the cecum. At present the proctitis has subsided, and yesterday's specimen was light brown in color, acid in reaction, had a slight increase in mucus, with still a small amount of pus and blood, and some yeast was present. The process, I judge, is still confined to the large intestine, more particularly in the cecum and ascending colon, which is the rule in these colitises when subsiding. Of course, one must be careful not to take a single characteristic and attempt to localize from that alone. For instance, one can change the alkaline stool of a colitis to a slightly acid stool by a long-continued high carbohydrate diet.

At present, as you see, the patient is still thin, though she has been gaining about 1½ pounds weekly for the last six weeks. She feels quite strong, is able to sit up in bed for several hours at a time, but cannot stand up. The cutaneous pigmentation is very marked. The blood-pressure is 115 systolic, 80 diastolic; the pulse ranges from 80 to 116. The temperature is normal, with an occasional rise to 99.2° F. The abdomen is flat, soft, and not tender anywhere. In short, the change from the profound prostration at entrance is quite marked.

The **treatment** of these non-specific infections of the gastro-intestinal tract is, of course, most difficult. To begin with the most important, namely, the diet. In type (*a*), the small intestine type, a high protein diet is very effective. We give meat, fish, gelatin and egg-albumen, and later the carbohydrates most easily absorbable, such as zwieback and gruels, together with small amounts of butter, tea, and broth. In large intestine disturbances, type (*c*), on the other hand, protein digestion is frequently disturbed, as evidenced by the putrefactive stools and their alkaline reaction. In such cases we increase the easily absorbed carbohydrates and diminish the proteins as far as possible. When, however, both are involved, as in this case at entrance, of course, the problem is still more difficult.

The following is an outline of dietary employed in this case.

On November 26th, for instance, she was given albumen-water 400 c.c., a mixture of sanatogen 0.6 per cent., and Robinson's Prepared Barley (flour) 0.6 per cent. in water, 450 c.c., malted milk (in water) 300 c.c., chicken broth, and twice unsweetened orange gelatin. The use of sanatogen or a similar protein preparation is very helpful in these cases. Solid food and especially meat is repulsive to them; milk is almost invariably poorly borne in small intestine disturbance, so that casein preparations are most valuable. Again, on December 8th, small amounts of cream were added to the sanatogen-barley flour mixture and to the malted milk, and 2 eggs were given besides the gelatin, a total of 960 calories. At this time the total urinary nitrogen was 8.25 gm. The fluid intake was 2600 c.c. All attempts to diminish the diarrhea by limiting the fluid intake were nullified by the intense thirst from the fever as well as the numerous stools.

During the following two weeks the occasional exacerbations of all the symptoms required our withdrawing practically all nourishment from time to time, so that it was deemed advisable to institute regular fast days at stated intervals, with, of course, quite intensive feeding in between. The influence of these fast days on both the temperature and diarrhea was quite marked, the bowel movements being two to three less frequent and the temperature being 1 to $1\frac{1}{2}$ degrees lower. The physical rest was also beneficial and the appetite improved on such days. Nothing but albumen-water, broth, tea, and water is given. At first every third day, at present every fifth day, is so designated and, of course, the interval will be lengthened.

On December 29th zwieback was added, first one slice, later more, later unsalted butter. On January 14th rice flour was added to the broth and also beef-juice, which she had previously refused.

These illustrate the best methods of increasing the carbohydrate portion. Cellulose and sugar are not well borne. Besides, fever patients dislike sweets. Starches without cellulose or with minutely divided cellulose and very thorougly cooked, are far superior. We therefore add flour to the various fluids,

as barley flour to the sanatogen mixture and rice flour to the broth. Another valuable type of carbohydrate in the later stages is the flour prepared from dried legumes, as peas and beans. They are, of course, very finely divided, and when cooked the starch is quite free. Their taste is pleasant.

In the third week of January meat was added, so that by the middle of February she was getting various sorts, as ham, sweetbreads, chicken and squab, but no scraped beef, as raw meat is not well borne. She was fed at two-hour intervals from 6 A. M to 8 P. M., and by the middle of February was getting over 2000 calories with 100 gm. protein, 119 fat, and 150 carbohydrate. While there is little evidence microscopically of food loss in the stool, still there must be considerable destruction, probably bacterial, in the intestinal tract, for instead of 12 to 15 gm. N. in the urine, a determination showed only 7.15 gm. She is, however, gaining in weight on an average of $1\frac{1}{2}$ pounds weekly, it will be remembered, in spite of the fast days and the intestinal destruction.

The remainder of the treatment has not been successful. For some time large doses of charcoal and chalk (9 or 10 teaspoonfuls daily) were given, with no result. Opium preparations by mouth were useless, and for a time morphin in $\frac{1}{8}$-grain doses were given three times daily hypodermically, but only during a very severe exacerbation. The danger of drug addiction was too great to continue it.

In this type of case, when the process is limited to the large bowel, two methods of applying medication are always to be considered—one per rectum, and one through an appendicostomy opening or artificial anus. I have found the use of the soluble preparations, both antiseptics and astringents, per rectum, such as quinin, silver nitrate, and even the milder astringents, as tannic acid, to result in such violent reactions as to be distinctly harmful. The use of the insoluble powders, as bismuth subnitrate or bismuth subgallate, is, of course, less irritating, but neither are they distinctly beneficial. In the subacute cases one can use 20 per cent. solutions of gelatin, given hot, or irrigation with several quarts of hot, normal salt solution. A few attempts

to irrigate with even small amounts of normal salt solution caused such a violent tenesmus in our patient that the attempt had to be abandoned.

All operative interference was rejected by this patient, who in the course of her five years' treatment has acquired quite decided ideas as to the efficacy of the various therapeutic measures. Neither an artificial anus nor an appendicostomy would have had any effect when she first entered the hospital, inasmuch as the small intestine was clearly involved in the process. At present her progress is so sufficiently sure, even though slow, that an irrigation from above, though advisable, is not imperative.

In general, one may say that an artificial anus with irrigation is effective in about 75 per cent. of cases, while medical treatment is effective in about 50 per cent. The main objection to an artificial anus in severe cases such as the present one is that strictures may develop when the colon is placed completely at rest, and we may then be obliged to leave the opening permanently, or run the risk of a recurrence by closing it.

CLINIC OF DR. JULIUS H. HESS

Cook County Hospital

THE CARE OF PREMATURE INFANTS

We will discuss another lesson in the need of preparedness as learned from our experience in the recent epidemics of influenza-pneumonia.

The little patient, Mary G., whom we are presenting this morning is but one of the many motherless infants born during the pandemics through which we have passed during the last two years. At no time previously have we seen such an influx of prematurely born infants into our wards at Cook County and Sarah Morris Hospitals. Our experience has not been the exception, but is the case with every institution handling obstetric cases or specializing in the care of infants. The greatest misfortune accompanying this influx of immature and not too infrequently, also, congenitally diseased infants has been the fact that so many of them had lost their mothers through pneumonia, and added to this disaster there had been a long delay in the institution of their proper feeding and care. In many instances no attempt had been made to maintain their body temperatures and to supply them with proper food of sufficient quantity.

In looking over the situation in Chicago we find not more than six institutions properly equipped to handle even a limited number of such infants. Such a unit must include a properly equipped nursery, special beds or a room in which the infant can be surrounded with warm, clean air of proper moisture and temperature, and be supplied with human milk; and, last, but equally important, a nurse of proper training and temperament. The Chicago situation is not unique; it is the story of practically every large city of the United States; in fact, the lack of proper facilities

for the care of premature infants is a deplorable situation which is almost universal.

Does the absence of proper hospital equipment minimize the life hopes for these infants? Is it possible to save a good percentage of them by good or even fair home care when human milk is available? We will attempt to study the care of these infants both from the standpoint of the complete hospital unit and in the home.

The term *premature* in the strictest sense of the word refers to those infants born before the end of the fortieth week of pregnancy, but in common usage it refers only to those infants who have undergone a gestation period of two hundred and sixty days or less, and so it may be understood that when the designation "premature" is used it refers to those infants born three weeks or more before the usual termination of pregnancy.

There is another class of infants that may be considered in practically the same category as the premature. These are the *weaklings*, the infants born possibly at term, or nearly so, yet who have suffered more or less severely during their intra-uterine existence from factors which interfered with their nutrition and, consequently, their development. They are classed as cases of *congenital disease* or *debility*.

It must be remembered that all infants born before the end of a normal term are born before the end of a full intra-uterine pregnancy, and consequently their organs are not fully developed, and as a result they must show certain bodily weaknesses and a lack of resistance to the traumas of extra-uterine life. This is the class of *immatures*.

This, however, is only a relative body weakness in the absence of inherited constitutional debility and malformations.

It is also a fact that the younger the fetus when leaving the uterus, the greater are the difficulties to be overcome in the carrying out of the required body functions necessary to life and, therefore, the lower its vitality.

In a study of premature and congenitally debilitated infants at least two factors in the life-history of the fetus must be considered:

1. The term of its intra-uterine life.

2. The state of its functional development at birth, as evidenced by the presence or absence of inherited disease.

Congenital debility is dependent upon constitutional influences in the parents and intercurrent disease during the term of pregnancy.

Classification.—For practical clinical purposes the group of infants comprising the premature and congenitally debilitated may be classified as follows:

1. Premature infants, with no pathologic changes.

2. Premature infants, with pathologic changes, due to:

(*a*) Constitutional disease and chronic infections in the parents.

(*b*) Maternal factors influencing the fetal nutrition, such as overwork, undernourishment, acute illnesses during pregnancy.

(*c*) Local conditions in the mother.

(*d*) Multiple pregnancies.

(*e*) Constitutional defects and congenital malformations in the fetus.

(*f*) Infants born to parents late in life.

3. Full-term infants with pathologic changes due to the same causes as those enumerated under 2.

Mary G., whom we are presenting this morning, belongs to the group of premature infants with no pathologic changes. Judging from its physical development, we believe it to have attained twenty-eight weeks of intra-uterine life. You will note that its general appearance is that of a small and puny infant, its weight 1020 grams. The skin is soft and pale and hangs in folds; the epidermis is thin and blood-vessels are easily seen. The adipose tissue is scant; the features are angular and the face looks old. The body is covered with lanugo, especially upon the extensor surfaces. The skull is ovoid; the fontanels are large and the sutures open. The ears are soft, small, and flattened against the skull. The nails are poorly developed and irregular. The infant lies in deep sleep and its cry is feeble and monotonous. Its movements are slow and there is a marked degree of muscle inertia. Its temperature is 97.4° F., with a

tendency to become even more subnormal except when the incubator bed, in which it lives, is maintained at a temperature below 85° F. Its respirations are shallow and approximate 50 per minute. The apex-beat is scarcely palpable.

The abdomen is large in proportion to the size of the remainder of the infant, and more especially distended over the region of the stomach. It has shown a tendency to marked constipation since birth, undoubtedly due to the poor muscular development of the intestinal and abdominal walls.

Its body measurements are as follows:

Circumference of the head	25 cm.
Circumference of the chest	21 "
Length of the body	36 "

This girl infant is the third born to this American mother, twenty years of age. The oldest, a boy, is two years of age; the second, a girl, is ten months of age; both were breast fed, are living and well. Due to an accident the last labor was precipitated during the late seventh or early eighth month of pregnancy. The mother's breasts have remained practically dry. Of interest to us are the following facts in this infant's history: She was born of a white American mother early in life, the third infant in a term of two years. Labor followed an accident, while she was in good health. There were no other direct prenatal factors tending to congenital weakness.

The determination of the exact *age of the infant prematurely born* is a matter of considerable difficulty. The information furnished by the mother as to the time of her last menstrual period, or as to the time when life was felt, gives an entirely insufficient approximation, and errors of a month or even more are not rare. The weight of the infant is of uncertain value, as an infant of 1500 grams weight may be the product of a pregnancy of seven months in a healthy woman, while one of the same or less weight may be eight months' offspring of an albuminuric or syphilitic mother. The *body measurements* also vary materially. The degree of development of the osseous system is of great value in determining the anatomic develop-

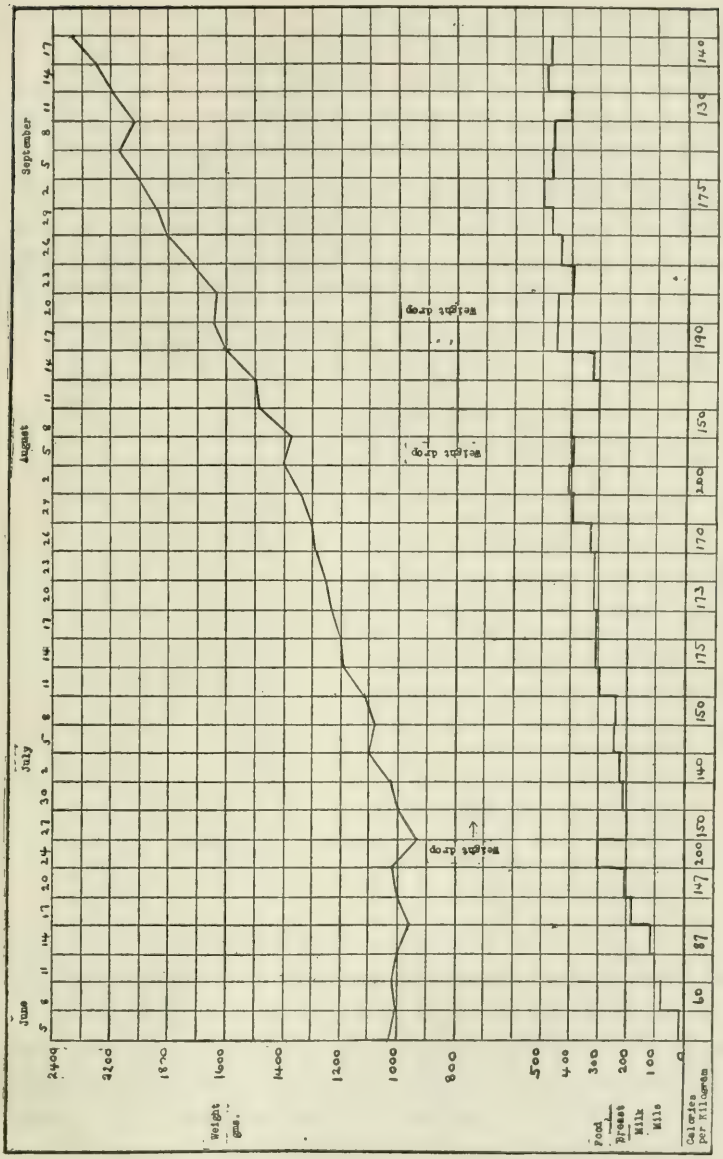

Fig. 262.—Chart I, Mary G.

ment and may be taken as a guide to physiologic development. Comparison of measurements in the same individual are of more

value than a consideration of one measurement alone. The circumference of the head is always greater than that of the chest in the normal premature. They also show relatively greater length of the upper half of the body as compared with the lower extremities, the latter growing with especial rapidity during the second half of the intra-uterine existence.

From a study of the various figures of body measurements, as taken from the literature, and such figures as we have taken from our own cases, we find that those quoted by Reiche may be considered as fair averages for the *normal prematurely born infant* which you will note in this lantern slide:

TABLE I

	Minimum.	Maximum.	Average.
Group 1: Weight, 800–1200 gm.			
Length of body	34 cm.	41 cm.	37.4 cm.
Circumference of chest	21 "	24.5 "	22.5 "
Circumference of head	24 "	29.5 "	26.8 "
Group 2: Weight, 1200–1500 gm.			
Length of body	37 cm.	45 cm.	41.6 cm.
Circumference of chest	22.5 "	27.5 "	24.8 "
Circumference of head	26 "	31 "	28.4 "
Group 3: Weight, 1500–2000 gm.			
Length of body	41 cm.	48.5 cm.	44.2 cm.
Circumference of chest	25 "	32.5 "	27.2 "
Circumference of head	27 "	32 "	30.3 "
Group 4: Weight, 2000–2500 gm.			
Length of body	41.5 cm.	49 cm.	46.5 cm.
Circumference of chest	26 "	30 "	28.4 "
Circumference of head	29 "	33.5 "	32.2 "

The figures show a gradual and steady increase of the weight and the chest and head measurements up to the time of maturity, when they should average 3200 grams in weight, 50½ cm. in length, with a chest circumference of 32.9 to 33.8 cm., and a head circumference of 34½ cm. We see in the eighth to the tenth month an abrupt rise of the curve of the chest circumference, the curve flattening somewhat only after the birth.

The weight depends upon the cause of the premature birth and upon the age of the child. Those born of mothers afflicted with nephritis, tuberculosis, or other wasting diseases, and infants

showing active syphilis, are usually considerably smaller than the same aged infants of healthy parents. Diseases and abnormal location of the placenta also restrict the growth of the fetus. The infant in placenta prævia is often undersized, even when born at term. Multiparity may predispose to undersize. The following tables, as seen in the lantern slides, represent the comparative weight and length studies of His, Oberwarth, and Ahlfeld, respectively:

TABLE II

His

Fetal age.	Weight.	Length.
16–20 weeks	250– 280 gm.	17–26 cm.
20–24 "	645–1000 "	28–34 "
24–28 "	1000–1220 "	35–38 "
28–32 "	1220–1600 "	39–43 "
32–36 "	1600–2500 "	46–48 "
36–40 "	2500–3100 "	48–50 "

Oberwarth

Fetal age.	Weight.	Length.
26 weeks	330–1041 gm.	28 –37 cm.
28 "	995–1408 "	36.3–37.5 "
30 "	707–1700 "	33.1–41.3 "
32 "	1868–1964 "	42 –42.7 "
34 "	1286–2213 "	39 –47 "
36 "	2424–2700 "	46.1–48 "

Ahlfeld

Fetal age.	Weight.	Length.
27 weeks	1140 gm.	36.3 cm.
29 "	1575 "	39.6 "
31 "	1975 "	42.7 "
33 "	2100 "	43.9 "
35 "	2750 "	47.3 "
37 "	2875 "	48.3 "

The **body temperature** shows a tendency to *hypothermia*, heat regulation being one of the least developed functions of the premature infant, the body temperature showing marked fluctuation. This is due to several factors. First, faulty heat regulation due to lack of development on the part of the nervous system. Second, loss of heat through radiation, in great part dependent upon lack of development of subcutaneous fat. The heat loss, therefore, is in great part proportional to the extent

of the surface of the body. Third, insufficient oxygen combustion due to the poorly developed respiratory center and the tendency toward asphyxia. Fourth, circulatory weakness. Fifth, insufficient heat production due to lack of food or improper metabolism.

Loss of body weight during the first days of life occurs almost constantly in premature infants, the percentage loss being greater in the premature than in the full-term infant, and, on the whole, they are much slower in regaining their birth weight. In the group of cases studied by me the average loss in the cases weighing between 1000 and 2000 grams was $10\frac{9}{10}$ per cent.

Most of our cases have regained their birth weight by the eighteenth to the twenty-first day, with a daily gain averaging from 12 to 40 grams after reaching their lowest weight, which is usually about the fifth day. Infants under 1500 grams may be considered as growing satisfactorily on an average of from 10 to 20 grams, and those from 1500 to 2000 grams when they are making a daily gain of from 15 to 30 grams after they have reached or passed their birth weight.

The table which we are now showing you, taken from the work of Ssytcheff, illustrates the comparative surface area in a premature and full-term infant:

Age.	Weight.	Surface area.	Surface area per kg. of weight.
Premature, four days old	1505 gm.	12.664 sq. cm.	841.4 sq. cm.
Newborn	2097 "	1476 "	704 "
Fifteen days old	2980 "	2129 "	711 "

Thus it will be seen that the larger the body volume (weight), the smaller the surface area relative to that weight.

TECHNIC OF THE GENERAL CARE

Asepsis.—The greater susceptibility of the prematures, especially of their less protected skin, demands even more painstaking observation of rules that hold good for newborn infants in general. These infants succumb more readily to infections and are much less resistant than are the full-term infants. Again, the frequently complicated feeding technic gives more opportunity

for affection of the digestive tract, so that in every form of indirect feeding careful attention to aseptic technic must be insisted upon. Also, the danger of infection of the respiratory passages is not to be underestimated.

Preservation of Body Temperature.—The preservation of temperature demands a very careful supervision, proper attention being paid to the thermolability and tendency to subnormal temperatures. The chief object in the preservation of the temperature is the prevention of excessive heat loss, which in itself may be a danger to the infant. This will also diminish the energy loss. Limitation of radiation may be accomplished in several ways. The infant may be surrounded by thick wrappings of material with poor heat conductivity, and by surrounding the infant with a heated air. A combination of these two methods is imperative, as all attempts to prevent heat loss entirely by keeping the infant in a heated air without attempts at insulating the body have resulted in failure and death. All attempts at maintaining a sufficient heat to keep up a body temperature approaching the normal in infants with a marked hypothermia without directly insulating the body result in a hyperpyrexia with symptoms resembling heat stroke.

The preservation of heat must be begun immediately after birth of the infant, preferably on the confinement bed itself, as the extent of the initial temperature drop is of no mean consequence to a premature infant. The umbilical cord should be severed early, the infant being received into warm towels or a blanket and immediately placed in a heated basket or incubator bed, which should be a part of the equipment of the delivery room. In the home hot-water bottles, a properly protected electric pad, or an improvised incubator will answer the purpose. It should be remembered that these infants are easily burned, and such burns are usually fatal. In small prematures for emergency use cotton-pack, completely enveloping the infant, except for the face, hands, and genito-anal region, answers very well. To the genital region and anus an easily changed small napkin of cotton or gauze combination may be applied. A jacket may be placed on the outside of the cotton

to hold it in place. Whenever the infant becomes soiled it is only necessary to change the napkin. This should not be neglected.

If a superheated bed is not available in emergency, the infant should further be placed in a coat made from a heavy woolen

Fig. 263.—To avoid the danger of fire from short circuits in electric heating pads a copper receptacle is used, 16 inches long, 13 inches wide, and 1¼ inches high, into which a 12 x 15 inch heating pad is laid. To allow of a maximum radiation from the lid or upper surface of the same the floor and sides are lined with asbestos sheeting, while the lid is not lined. The cord passes through a small rubber insulator at the side to prevent contact with the metal and injury to the cord.

This simple device can be used temporarily in wards and homes where better facilities for the care of this class of infants are lacking. It is to be placed in the bottom of a basket or crib, under the mattress or pillow.

blanket, or cotton combination, which can be fastened about the body loosely by bandages or safety-pins. The greatest disadvantage of such a dress is the limitation of body movements, which is of considerable importance even in these infants.

In a well-equipped station several sets of cloths should be provided. These should be kept sterilized in packets. Whenever an infant is to be raised in an open incubator bed, it should be provided with woolen garments, consisting of light-weight woolen bands of small size. Small woolen undershirts and a woolen bag, with an attached head-piece, with a slit over the upper part in front to allow passing over the head. The bag should be open at the bottom to allow of its being raised for

Fig. 264.—Water-jacketed infant bed.

changing of napkins, dressing cord, and general care of the infant. Woolen stockings should be used. Therefore, for use in an open bed whenever possible, all clothes except the napkins should be of wool.

In practice we depend mainly upon two methods of *maintaining external heat:* first, by the use of a well-insulated copper jacket with its floor and inner walls covered by asbestos sheeting, within which is placed an electric heating pad. This is now

placed in the bottom of a crib which has been well padded and lined with cotton or woolen pads. Such a protected electric pad is illustrated in Fig. 263.

In our incubator station we have a battery of water-jacketed beds, such as you see in Figs. 264 and 265. These beds fulfil the following needs of the infant: first, *safety*. The maximum

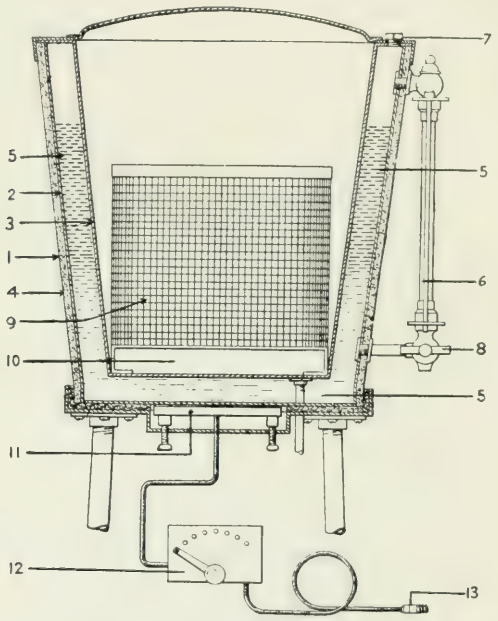

Fig. 265.—1, Copper wall covering asbestos layer; 2, asbestos layer insulating water-jacket; 3, 4, copper walls covering water-jacket; 5, water surrounding side and floor of bed; 6, water glass; 7, funnel for filling jacket; 8, cock for emptying jacket; 9, removable crib; 10, air space underneath crib; 11, electric heating plate; 12, rheostat; 13, electric plug.

temperature which can be attained within the bed is about 110° F. when the lid and canopy are in place with a room temperature of 70° F. While such a temperature would be injurious if maintained for a long period of time, such surroundings, if temporary, can cause but little injury. Second, *simplicity of operation*. It requires practically no attention unless there are extreme ranges of temperature within the ward, since the as-

bestos insulation prevents radiation from the outer surface of the bed and the heater holds the water at a constant temperature. Third, *ventilation*. This apparatus insures the baby of an adequate supply of fresh air if placed in a properly ventilated room. Fourth, *humidity* is maintained at practically the same degree as the surrounding air because of the almost constant change of air within the bed. Fifth, it is *easily cleaned and disinfected.*

The temperature within the bed should be read and charted at six-hour intervals, best at 6 A. M., 12 M., and 6 and 12 P. M., as the most likely times for maximum changes in the ward temperature.

CHART II

TEMPERATURE CHART FOR INCUBATOR BED

Day of entrance.....Case No.....

Name..... Dr.....

Date.	6 A. M.	12 M.	6 P. M.	12 P. M.

The construction of the bed is such that it is intended for use in an ordinary ward or room, giving the infant the advantage of the most perfect room ventilation.

The Nursing Staff.—The selection of a personnel for the nursing staff of a unit established for the care of premature infants requires great care. Nurses having the care must be intensely interested in their work. They must be willing to make the necessary sacrifices in passing through the critical stages in the life of such an infant. They must, at all times, be prepared to meet emergencies of asphyxia and to counteract the spells of cyanosis. These two factors in themselves require

almost constant diligence, otherwise the work of previous days will go unrewarded. They must use good judgment to prevent over- and underfeeding, as to a very great extent the size of the individual meal will be dependent upon the physical condition of the infant at the time of feeding. In no other class of patients is it so necessary to change or modify previous orders for diet. The nurse must know the indications for and the methods of administering catheter feedings, colonic flushing, tubbing, and the application of artificial respiration.

In our hospital wards we have found the constant changing of nurses, as is so frequently the case in meeting the curriculum for nurses' training in general hospitals, to be of the greatest disadvantage. Far better results are obtained when the nurse in charge has under her care assistants who need not necessarily be nurses in training, but preferably young women who are especially preparing themselves for the care of young infants, and who can be relied upon to stay in the station for long periods of time. Such women become expert in the handling of these infants, can frequently feed them with a minimum of excitement of their reflexes, and soon learn to bathe and give them their exercise and massage, which is so essential to every infant in order to prevent "hospitalization." The ideal nursing staff for such a station is, therefore, one consisting of a well-trained supervising nurse and a corps of assistants desiring this training, and who are willing to remain in this service for a long period of time.

Removal of infants from their bed should be practised with forethought. The small infants should, so far as possible, be manipulated only upon a definite indication. This is, first, for cleanliness, including bathing; second, exercise, including gentle massage, all of which should be performed before feeding. In most instances the food, when administered other than by catheter, can be given without removing the baby from the bed. Catheter feeding in infants not subject to cyanotic spells can often be performed to advantage without removal from the bed. When cyanosis is present or easily precipitated the infant should be removed from the bed during feeding.

In preparing the infant for *permanent removal* from the heated bed the surrounding temperature should be gradually lessened until the ordinary room temperature is reached.

The **nursery** should be a room independent of the station in which the superheated beds are kept. It should be provided with double windows, a good system of heating, and must be kept immaculately clean, good ventilation and general cleanliness being essential. All infants are to be bathed in this room, and whenever possible it should be provided with either a bathing board or water-jacket provided for the same purpose. These are covered with a sterile towel for each infant. The older infants are also to be fed in this room. It must also be provided with proper linen closets, a simple dressing table, a scale, a hygrometer, and thermometers which register not only the present temperature but also the extremes for the twenty-four hours. This latter is one of the best methods of testing an efficient nursery. A time clock should also be provided and all feedings registered by this method, so that the supervisor may have a constant check on the activities of her assistants.

The general hygiene and care of the infant in the nursery is second only in importance to an ample supply of human milk and a maintenance of the body temperature of the infant.

Home Care.—In the home care of these infants the same rules for hygiene, maintenance of body temperature, breast feeding, and daily routine must be maintained as suggested for their hospital care. Whenever possible, two rooms should be set aside for the infant's use; one equipped as a nursery, with similar furnishings as described for the hospital nursery. The second room should be used for sleeping quarters and must be supplied with a heated bed. This room must be capable of being well ventilated and at the same time well heated. In both these rooms all draperies and unnecessary furniture must be removed. The nurse's quarters must be in close proximity to the sleeping quarters of the infant, as must also the mother's or wet nurse's room. The nurse selected must be experienced in the feeding and handling of such infants and must be tireless in her efforts to prevent complications. Her temperament should

be such as to permit of overcoming the mother's anxiety, and of controlling the habits of the wet-nurse if one is employed. Breast milk must be obtained from an outside source until the mother can supply it. *All visitors must be excluded.*

Methods of Feeding.—It is necessary to consider these infants as belonging to two large groups: first, those able to nurse at the breast; and second, those too weak to nurse at the breast.

Infants Nursing at the Breast.—This presupposes that the infant has the proper physical development to withdraw milk from the human breast in the presence of an abundant supply and well-developed nipples. Such an infant may be placed on the breast two or three times during the last half of the first day after the circulatory and respiratory functions are well established. Following the first day it should be placed at the mother's breast regularly for two- or three-minute periods at three- or four-hour intervals, even though the breast contains little milk. Following these attempts at nursing, food should be supplied from another mother or a wet-nurse. In the hospital it is our custom to give these feedings by hand; in private practice the infant may be placed to the wet-nurse's breast, one of the breasts being set aside for this purpose, and if there is a difference in the breasts, the better one selected. Not infrequently great assistance may be given the infant in securing its milk by one of two methods: Either by expressing the milk directly into the baby's mouth, or by placing the wet-nurse's baby on the opposite breast, which reflexly stimulates the flow of milk into the opposite breast, thereby assisting the weak infant in obtaining its food. *Overfeeding* becomes a danger in this direct application of the infant to the breast, and weighing before and after nursing should be practised. Whenever possible this is the best method of getting the food to the baby, as it prevents contamination of the milk, stimulates the breasts, and develops the baby's independence as well as his sucking muscles. *Underfeeding* is an even greater danger, and here again the infant must be weighed before and after feeding to ascertain the amount of food taken. If insufficient, further food can be supplied by hand feeding.

Infants Too Weak to Nurse at the Breast.—In this group of infants careless exposure must be avoided, and this is best accomplished so far as possible by feeding them without removal from the bed. The inability to nurse may be due to improper development of the nursing center, lack of co-ordination on the part of the pharyngeal muscles and tongue. This is usually made evident by a return flow of milk from the mouth. Again, the infant may be too weak to nurse, or it may not have learned to suck, or vomiting, and again, cyanosis may prevent its feeding properly. In this group of infants we may, of necessity, resort to one of several procedures: the use of a fruit *spoon* or, better, a large size *medicine-dropper*. In those infants who can assist themselves, a small *nursing bottle* provided with the small nipples commonly sold on doll nursing bottles, which can usually be obtained of proper quality. Such a nipple can be made by perforating the rubber bulb of a medicine-dropper. The *Breck feeder* in the original, or a modification which can be made by flanging the ends of a urethral syringe, using a rubber finger-cot on one end and a small nipple on the other, will usually suffice. Direct expression of milk into the infant's mouth has proved one of the most valuable expedients in our hands. *Catheter* feeding is the simplest and best method of procedure in the smaller infants if carefully practised by an experienced nurse. A catheter (No. 14 French) about 14 inches in length may be attached to a small funnel, graduated glass tube, or, in case of emergency, the glass barrel of a small syringe may be used. All food should be carefully measured and administered slowly with a minimum elevation required to obtain a free flow of the milk. The infant should be upon its back on a flat surface with the head either in the median line or turned to the right. The passage of the catheter is usually effected without difficulty by passing it in the midline to the pharynx, gradually pushing it into the esophagus. The poorly developed reflexes rarely result in retching. The distance to which the catheter is to be passed is of great importance when we consider that this procedure must be repeated at least four to six times daily over a considerable period of time. It has been our rule to measure the distance

from the bridge of the nose to the tip of the ensiform cartilage, which is usually in the neighborhood of 10 cm. The catheter is marked at this point, and is passed about 1 cm. short of this distance. It reaches the lower end of the esophagus and the food will flow through the patent cardia, and we thereby avoid irritating the gastric mucosa. The milk is now allowed to flow into the stomach slowly, the funnel being raised only slightly above the level of the body. After the feeding the catheter is firmly compressed to avoid spilling milk into the pharynx during its removal.

The Number of Feedings.—This will, of necessity, depend in many instances upon the question of catheter versus other methods of feeding. Larger infants fed by catheter can often be given sufficient food at four-hour intervals to meet their needs. In small infants fed by a dropper, bottle, or other methods we have experienced great difficulty in administering a sufficient quantity of food by the long-interval feeding. As the attendants in charge are frequently not to be trusted with the catheter feeding, the short interval of feeding must be resorted to. For this purpose we have grouped our infants into two classes, those weighing under 1500 grams and those weighing above this figure. These figures are arbitrary and will not require rigid adherence. The classification is based on the tendency of the smaller infants to become exhausted when the feedings are too long continued. The smaller infants are fed at two-hour intervals during the day and three hours at night. The larger, on the three-hour basis. It must be remembered that all feedings are dependent on the general development of the infant in relation to its digestion and metabolism and its ability to retain the food administered, as well as attendant complications to feeding, such as asphyxia, cyanosis, and gastric distention.

When should *regular feeding* be started? This is a question of great importance to these infants because of the tendency to develop acute inanition. Therefore a regular feeding régime must be started early. Human milk is essential to a low mortality. As little can be expected from the mother for several days,

it becomes necessary to obtain the limited supply necessary from another mother, preferably a wet nurse, if for any reason it is unlikely that the mother may be depended upon, either because of illness or of local breast conditions.

During the *first day* it is our custom to withhold milk for at least twelve hours until the respiratory and circulatory functions are well established. During the second twelve hours one to three feedings of breast milk may be started if the infant's condition warrants. The *second to the tenth days* may be grouped together as the second feeding period for practical feeding purposes.

From the second day they should be fed regularly, day and night, the number and time of feedings depending to a great extent on whether the food be given with or without the use of a catheter; second, upon the gastric capacity; third, upon the infant's general condition.

Further, fluids, preferably inert, such as water or 1 per cent. lactose solution, are administered to compensate for the loss of body fluids through the kidneys, bowels, and skin. The infant requires about one-sixth of its body weight of water, inclusive of that contained in the milk, in twenty-four hours. Such quantities, however, should not be attempted on the first days. The early feedings must necessarily be small, and the increases gradual.

Each infant must be fed individually, as it is impossible to formulate definite rules for feeding, at least during the first ten days. First, we must have a definite idea of the minimum food requirements for life; second, that necessary to maintain at least a stationary weight, and, lastly, the amount of food needed to meet the requirements for growth and development. Approximately one-sixth of the body weight of fluids and caloric food value of 70 calories per kilo are required to maintain life. Little can be expected in the way of weight increase until 90 calories are reached, and depending on their weight, body surface, and physiologic development, their later needs will approximate 110 to 140 calories per kilogram body weight.

Infants, to fulfil all their needs, will therefore require from

140 to 200 mils. of breast milk per kilo, or about one-seventh to one-fifth of their body weight daily. They can, however, maintain life on 100 mils. and hold their weight in most cases on 130 mils. per kilo.

Beginning (in most cases by the second day) with 20 to 40 mils. of human milk per kilogram of body weight, the quantity may be increased by 8 to 15 mils. daily per kilogram until, usually by the tenth day, feedings averaging from 80 to 140 mils. per kilogram can be fed. The latter figure usually holds true only in those weighing over 1500 grams.

These feedings should, as rapidly as possible, be supplemented by water or sugar-water by mouth, or saline by rectum, to meet the required 140 to 200 mils. per kilogram of fluids required daily.

After the tenth day in larger infants the food can be increased more rapidly, until from 130 to 200 mils. per kilo are fed, the methods of giving the food, as well as its frequency, being dependent on the general development of the infant.

The size of individual feedings will vary with the method of feeding. When *catheter* fed, six feedings a day are given, with an average of from 4 to 6 mils. per feeding during the second day. The feedings are now increased daily by an average of from 1 or 2 mils. per feeding. When feeding from the *bottle* or by dropper, smaller feedings are usually given more frequently, usually from eight to ten daily, beginning with 2 to 4 mils., and increasing by 1 mil. per feeding on each succeeding day, until 130 to 150 mils. per kilogram per day is reached.

The diet of a premature infant making a satisfactory gain in weight should not be changed arbitrarily without a well-defined indication.

Daily Gains.—These are not necessarily in proportion to the changing quantity of milk administered, as many factors, such as condition of the bowels, quantity of urine passed, temperature of the infant's surroundings and numerous other factors, will necessarily influence the weight.

An average daily gain greater than 20 grams is unusual when the infant's food is limited to one-fifth of its body weight. An

average of from 10 to 20 grams daily gain can in most instances be considered satisfactory.

ARTIFICIAL FEEDING

There can be no comparison between the results to be expected in feeding premature infants on human milk and those to be obtained with artificial food. Therefore, if it becomes necessary to resort to artificial feeding, the selection of a food, its preparation, and its adaptation to the infant must all be given the most painstaking consideration. Many varieties of artificial diet have been suggested by many different authors, such as simple milk dilutions, cream and top-milk mixtures, skimmed milk and buttermilk preparations, malt soup preparations, condensed and evaporated milk, etc. The results with these various diets are to a great degree dependent upon the physician's intimate understanding and directions for the use of the individual food.

Quantity of Food.—It must be remembered that the figures quoted for the feeding on breast milk are the maximum that can be assimilated, and are excessive quantities for artificial feeding in the first weeks of life because of greater difficulty in the digestion of cow's milk. These infants when artificially fed must at all times be closely watched for evidences of overfeeding, and the first evidence of digestive disturbances or of intercurrent infections should lead to the feeding of human milk whenever possible.

From the foregoing statement it is quite evident that smaller and slower weight increase may be expected of the artificially fed.

Quality of Food.—As previously stated, opinions vary greatly as to the best food for artificial diet. Most clinicians have obtained the best results with the feeding of low fat mixtures. Boiled milk, skimmed milk, and buttermilk, with carbohydrates added, are among the best. Such a mixture can be made by adding 10 grams of flour and 40 grams of sugar to 1000 mils. of skimmed milk or buttermilk. Such a mixture can be strengthened at will by the increase of the flour and sugar content. We have found the predigestion of boiled skimmed milk, by the addi-

tion of pepsin or rennet (chymogen), assures the infant's stomach of a fine, flocculent curd, which is about the size of that of human milk. In beginning feedings with the above preparation they are usually dilated with 3 parts of water and increases in quality made as indicated, and the quantity increased as in the feeding of human milk. In feeding with the predigested milk, ½ per cent of lactose should be added during the first few days, and the amount gradually increased to 3 per cent.

Mixed Feeding.—When human milk, even though in small quantities, is available, it should form the basis of the diet, and cow's milk should be supplemental.

DISEASES PECULIAR TO PREMATURE INFANTS

In the short time available for the discussion of the pathologic condition of this class of infants we will not attempt to speak of the effect of acute and chronic conditions in the parents, leaving these for a more general survey in speaking of the prognosis. There are, however, a group of conditions peculiar to the prematurely born, among which the following are the most frequent:

Hypothermia, with its attendant dangers and the means of overcoming them have been discussed.

Hyperpyrexia is a frequent condition, and is not uncommonly seen in the absence of infection, when it is mainly due to one of three causes: intracranial hemorrhage, starvation, and excessive external heat. Starvation must necessarily be avoided, as these infants react very poorly to insufficient feeding, thereby increasing the danger of secondary infection.

Because of the instability of the nervous system they react in the extreme to their surroundings, and when intense external heat in the presence of a low humidity is applied, hyperpyrexia is not an uncommon result. The temperature usually drops rapidly with a change in the surroundings.

Cyanotic attacks may be precipitated by several factors. They are not uncommon immediately after birth, owing to poor central response to external stimuli and the needs for respiration. Pulmonary atelectasis, either temporary or permanent, as well as circulatory disturbances, which latter may be due to a weak

heart action or a tendency to continuation of the fetal circulation through the ductus botalli and foramen ovale, resulting in a minimum pulmonary circulation, are frequent causes. Chilling of the body, with dilatation of the pulmonary and mesenteric reservoirs, may precipitate attacks. Again, starvation, with its consequent exhaustion, overfeeding and too rapid feeding, with subsequent gastric distention, are often direct factors. Aspiration of mucus early or food later may result in cyanosis. To properly treat these attacks the cause must be recognized. The early attacks, if not due to aspiration or refrigeration, are most commonly central or circulatory. During the cyanotic attacks the infants apparently forget to breathe and, peculiarly enough, when life seems almost extinct, will again start to breathe spontaneously, when the blood becomes sufficiently laden with carbon dioxid to stimulate the respiratory center. Gentle but regular artificial respiration, warm baths, oxygen, and a drop or two of aromatic spirits of ammonia are the best means for overcoming the individual attacks. The cases due to gastric distention are always a source of great anxiety because of the added danger of starvation. These infants are best fed either in their bed or on a flat table. When catheter fed, the head should be on a plane with the body and the food given slowly. They should be placed on the right side after feeding. When the spells develop in infants fed either directly at the breast or from a bottle, they are frequently relieved by slow catheter feeding. Infants suffering from these attacks must be under constant observation, and preparation for treatment must be complete.

Aspiration of mucus or food is best relieved by inverting the infant, gently swabbing the throat with a finger protected by cotton, or, in extreme cases, by catheterization.

Atelectasis pulmonum, if extreme, is almost invariably fatal sooner or later. It must not be confused with congenital heart disease or diaphragmatic hernia. A most careful physical examination assisted by radiographic studies will often be necessary to make a diagnosis. The treatment consists in the early application of artificial respiration, gentle rubbing and spanking, and warm baths. Inflation with a tracheal catheter may be

necessary. In the latter procedure the greatest care is necessary to prevent traumatizing the respiratory passage and, again, to prevent rupture of the alveoli.

Convulsions may be due to intracranial hemorrhage, asphyxia, gastro-intestinal disturbances, hyperpyrexia, and infection. It need only be stated that the diagnosis of the etiologic factors alone give a rational basis for therapeutic procedure.

Gastric and **intestinal indigestion,** with resulting distention, may require lavage or intestinal irrigation. Cathartics must be administered with great forethought. Small doses of castor oil are best.

Infections may involve any and all parts of the body. Among the most distressing are those of the nasal passages and mouth, because of their interference with respiration and nutrition, as well as their being a source of secondary invasion. Infections of the respiratory, gastro-intestinal, and genito-urinary tracts are common, and only the most painstaking asepsis will lessen their frequency. These must include clean, warm, fresh air, proper food, and feeding technic.

Rachitis, spasmophilia, and **anemia** are among the most frequent secondary diseases. They are overcome by the institution of a proper diet, including phosphorized cod-liver oil and the early administration of food rich in iron and iron medication.

Hydrocephalus.—The rapidly developing distention of the fontanels, so frequently seen in the second half year of life, is usually due to the rapid development of the brain, and usually disappears spontaneously without after-effect in the absence of congenital syphilis.

Spastic paraplegias and **diplegias** are more common in the premature than in the full-term infant and require special attention.

PROGNOSIS

The prognosis of prematurity depends partially upon the degree of immaturity, but even more upon the debility of its constitutional sense. Experience teaches us that it is impossible

to base our prognosis as to life upon definite minimal figures of weight, length of body, or other measurements.

Baby H., with a birth weight of 850 grams, reaching a minimum of 645 grams on the tenth day, left the obstetric service of Dr. L. E. Frankenthal at Michael Reese Hospital weighing 1640 grams when five months of age.

Our most interesting experience was with Greek twins, weighing 690 and 740 grams at birth, with a minimum of 490 and 509 grams on the eighteenth and twentieth days. They lived to seventy-one and seventy-two days respectively.

The prognosis is directly dependent upon the degree of proper care immediately after birth and the ability to meet the early emergencies of feeding and complications. If one succeeds in keeping the initial temperature decline within certain limits, further protecting the infant from subnormal temperatures, much is gained. The early institution of feeding with breast milk lowers mortality. If the general reactivity is low and the food intake imperfect, respiratory disturbances, subnormal temperature, and sclerema are prone to occur, and they are indications of serious debility. Debilitated infants of higher birth weight and conception age often have a less favorable prognosis than smaller but stronger infants not suffering from congenital debility.

CLINIC OF DR. FRANK WRIGHT

MERCY HOSPITAL

HYPERTENSION IN A WOMAN AT THE MENOPAUSE

THE frequency with which you meet with the finding of arterial hypertension prompts me to present this patient for your consideration today. I have seen her at frequent intervals for the past five years, and previous to that time less frequently for four and a half years.

Mrs. L. is now fifty years old, and gives this history: Childhood and adolescence were free from accident and infection with the exception of measles; menstruation began at thirteen, was normal and regular; marriage at twenty, which resulted in seven pregnancies in fifteen years, with delivery at term in each instance of a living child, two of whom died, one at three and one at eight months, of gastro-intestinal disease. There were no complications during any of the pregnancies and postpartum periods were uneventful. She says she had her children "easy." Bringing up the five living children was another matter; she had the entire care of her own household, which required strict financial strategy, and the burden of looking after her aged parents, who lived a few doors away. There seemed to be a never-ending procession of contagious diseases and respiratory infections among the children, but the mother remained normal, explaining that she never had time to be sick until she was forty-one. At this time a vague epigastric distress with appearance of constipation for the first time soon gave way to intermittent attacks of colic and vomiting, with tenderness over the gall-bladder area. Any doubt as to the diagnosis of cholelithiasis was removed six months later by an unusually long period of recurrent colic, jaundice for about ten days, the search in the stool,

which was acholic for about three days, for the elusive gallstone, which was not found. However, there has been no repetition of the attacks, and for four years there was no hint of abdominal discomfort except a tendency to constipation.

At the age of forty-three she gave a greater share of her time for six months to the care of her father, who died, after being hemiplegic for that period of time. Two months later the care of the mother during pneumonia devolved upon her. About this time she consulted me to see if there were any findings similar to those of her father which might be inherited, although the only complaints were of lassitude, weakness on exertion, and insomnia. The patient was overweight, 188 pounds with a height of 61 inches; there was a moderate anemia, 77 per cent. hemoglobin, and the blood-pressure was 140 systolic, 84 diastolic. Two specimens of urine were of 1016 and 1021 specific gravity and quite free from all pathologic constituents.

Knowing the experience she had undergone, I used every means to reassure her and relieve her of the fear that she was following in the footsteps of the father, whom she said she resembled.

Two years later, the patient being then forty-five, she appeared with the first symptom which brings her to our attention today. For three months she had noted a very free menstrual flow which amounted in the period then completed to a menorrhagia, with which there had been associated a violent headache several days before and continuing through the first day of menstruation, but ceasing when the flow was well established. With this was associated some dizziness, and between periods a feeling of unrest, emotional instability, and an urge to be "doing something," just what she could not say. The record at this period contains the first notation of a blood-pressure above normal, the reading being 176–114, and one week later 168–110. The patient at this time weighed 180 pounds; the heart gave no evidence of gross enlargement, there was some accentuation of the aortic second sound, but no murmurs and no irregularities. Night and morning urine, in separate bottles, examined four times in a ten-day period, ranged in specific

gravity from 1014 to 1018, contained albumin but once, and then showed some pus corpuscles which could have come from admixed vaginal secretion; no casts were found and cylindroids showed but twice; no other pathologic constituents were present.

The treatment comprised a cessation of all activity for two weeks, a light diet free from meat and extractives, small doses of bromids to secure relaxation, and bowel regulation, obtained principally by fruits and coarse breads. About this time Parke, Davis & Company had furnished me with corpus luteum extract in ampoule form for intramuscular administration; this I had been administering principally to patients upon whom an artificial menopause had been precipitated by the use of the deep Roentgen-ray treatment of fibroids. This was used twice each month, seven and three days previous to the expected onset of the menstrual period. Symptomatic relief followed for several months, but the blood-pressure varied each time taken, readings of 142–96 being followed, in one instance, a week later by a rise to 164–100. A change in the financial status of the family, with removal of residence from a noisy commercial street having a carline to a quiet place near Douglas Park, did much to promote the welfare of the patient, and for two years there were the usual fluctuations seen in such conditions. During this period it was pretty well established that there was a definite accentuation of headache, dizziness, and blood-pressure reading periodically, and usually preceding the menstruation, although in two or three instances a comparatively quiet monthly period was followed by exacerbation of symptoms; the corpus luteum by mouth or intramuscularly tended to relieve symptoms, often with depression of blood-pressure, but hastened menstruation, until finally each injection was followed by the appearance of the catamenia the same night or following day, and for that reason had to be discontinued. Later, ovarian substance was used with somewhat better results as regards induction of the flow. Rest just before and during the period was of great advantage. A course of cabinet baths resulted favorably. Any argument or altercation produced symptoms at once, and the patient remarked that she could "go crazy over a nickel."

For the greater part, the condition remained at a standstill; diet was well observed, food dissipation being more apt to produce abdominal discomfort and constipation than headache. The urine failed to show either albumin or casts, mycturia not present. The blood-pressure was usually between 160–180 systolic and 100–110 diastolic.

With the onset of the war and the worry incident to the registration and draft the physical condition of the patient changed at once. The pressure rose to 210–110; then a reading of 274–120 was recorded. Her eldest son was drafted and left early for France. The fear regarding his safety was not assuaged by the awakening to the fact that he produced a good percentage of the family income. Later, the second son was sent to a training camp, and his contribution to the support of the home removed. The patient was compelled to do more work than formerly, and conditions in every way were less favorable. Attempts to release the boys from service, when unsuccessful, precipitated attacks of vertigo, palpitation, violent and protracted headaches, and pains in the back. A general hyperesthesia developed. The menstruation increased in frequency until a metrorrhagia was added. The pressure again rose until 280–136 was recorded. The weight decreased to 162 pounds and a hemoglobin of 64 per cent. was determined. Rest in bed was required a greater share of the time.

With various remissions and exacerbations the patient continued at home until January of this year, when the picture again changed. With each menstrual period came a headache more profound than formerly, with pain extending down the neck and between the shoulders, which was agonizing. A sudden tachycardia would develop, followed by syncope, the patient becoming cyanose, with a cold sweat. There was marked substernal pain. The pulse-pressure rose, and the heart area was found to be enlarged. After these symptoms had recurred the patient entered Mercy Hospital for observation, that plans might be formulated for her relief.

Examination upon entrance showed a weight of 156 pounds, temperature 98.6° F., slight pyorrhea, very slight findings of

arteriosclerotic retinitis, as evidenced by tortuous small arteries, with some interruptions of the light streak, and distortion of the veins at the arterial crossings. There were no hemorrhages in the retinæ and no signs of albuminuric retinitis. The thyroid was normal. The heart showed an enlargement of precordial dulness extending 13 cm. to the left of the midline, there was a systolic murmur at the base and over the aorta, with an accentuated aortic second sound. The pulse was 100. The lungs were normal. The liver was barely palpable, but slightly tender; the gall-bladder was not palpable. The lower pole of the right kidney was palpable and somewhat movable.

Vaginal examination disclosed a lacerated, unrepaired perineum, laceration of the cervix with a corpus freely movable, slightly enlarged, hard, with easily palpable vessels, which felt sclerotic. There were a few external hemorrhoids. The reflexes were exaggerated.

The blood count showed hemoglobin of 70 per cent.; erythrocytes 4,600,000, leukocytes 9200. Blood-pressure was 224–102. The serum Wassermann reaction was negative.

A lumbar puncture with removal of 10 c.c. of cerebrospinal fluid was followed by relief of headache; this was later repeated, 15 c.c. being abstracted. The Wassermann reaction on the fluid was negative, cell count 8, tests for globulin negative, the urea nitrogen in the fluid being 0.1 mgm. per cubic centimeter. Dextrose was negative.

Chemical examination of the blood gave a total non-protein nitrogen 28, urea nitrogen 18, creatinin 3 mgm. per 100 c.c., blood dextrose 0.20 per cent., CO_2 combining power (van Slyke) 58.

None of these findings can be considered abnormal and were not taken as any indication of retention as would usually be found in interstitial disease of the kidney. The creatinin may be said to be a trifle over normal, but taken alone can mean but little. The high figure for dextrose I find frequently in hypertension cases. It is quite possible that other pressor principles have the power of increasing the sugar content of the blood, just as adrenalin can do when injected.

Renal examinations gave negative results, two single specimens 1012 and 1015 specific gravity, no albumin or casts. Three twenty-four-hour specimens ranged from 1000 to 1600 c.c. and from 1015 to 1017 specific gravity, and were likewise negative except for an occasional hyaline cast.

Phenolsulphonephthalein test showed appearance of dye in ten minutes, with 55 per cent. excreted in two hours.

The two-hour test diet for renal function gave a prompt elimination of fluid and nitrogen, small night quantity, a range of gravity of 15, and a concentration of nitrogen to approximately 1 per cent. in one of the specimens. Salt elimination was slow, the amount passed at night being 20 per cent. larger than that passed during the day. The concentration, however, reached 0.8 per cent.

The kidneys, therefore, appeared to functionate well, and in neither urine examined, functional kidney tests, nor blood examination were there any findings which would make for a diagnosis of chronic interstitial nephritis.

The problem of what to do became somewhat less complicated after all the findings were correlated. Because of the fact that relief from headache followed the first lumbar puncture, a second was done, after which, and coupled with the rest in bed, the blood-pressure fell to 188–124, while precordial distress persisted and led to the administration of 20 per cent. benzyl benzoate in 30-drop doses three times a day, with subsequent relief from the anginal pains. A purin-free diet was maintained and the salt reduced to a comfortable minimum from the gustatory standpoint, the latter based on the lag in the ability of the kidney to excrete it.

The distinct relationship between menstruation and uprise of symptoms prompted me to urge strongly the induction of menopause by the use of radium. This was refused at first, but after leaving the hospital and having a repetition of the stormy symptoms once more, the patient re-entered two weeks later and, after a couple of days' rest, 135 mgm. of radium sulphate was inserted into the uterine cavity under aseptic precautions, and allowed to remain for twenty-two hours. There was little

reaction, the patient left a week later and has not menstruated since.

The pressure the last time it was taken was 172–114, and a condition of comparative comfort has followed. She still maintains her diet, restricted as to quantity, purins and salt, rests a good share of the day, but is able to get out of doors and walk ten or twelve blocks.

In summing up the history and findings one is impressed with the following facts:

(1) That a high pressure was tolerated for some time by this woman.

(2) That nervous influences distinctly affected many of the symptoms.

(3) That arterial change developed gradually, as shown in the eye, the coronary arteries, and possibly the abdominal aorta and in the uterus.

(4) That the kidney partook but slightly of the difficulty.

(5) That the endocrine influence was here a factor, but probably only one of several.

(6) That one may be warranted in causing a cessation of menstruation if progression of symptoms requires it.

CLINIC OF DR. MILTON PORTIS

ST. LUKE'S HOSPITAL

TWO CASES OF SYPHILIS OF THE LIVER SIMULATING GALL-STONES

ALL of the patients that I am going to present to you this morning are from my private service in St. Luke's Hospital. The first 2 cases are of syphilis of the liver, which simulate gall-stones very closely. The first one was first observed on April 23, 1919, and I am now presenting him to you on his return for further treatment.

Case I.—This gentleman, Mr. E., aged fifty-four years, gave the following history when he first consulted me last year: He complained of mild distress in the region of the gall-bladder at various times for the past six or seven years, but the distress was never severe enough to interfere with his work, although at times he suffered with indigestion for a period of several days. Seven days ago a sudden, severe pain began in the right upper abdomen, and this has been constant both during the day and night. There has been no vomiting, but the patient has been nauseated at times. The pain is worse on sitting still, but is not increased by taking food. The pain at times radiates to the back and up to the right shoulder, but it does not remain constantly except in the region of the gall-bladder. He has not had much appetite, his bowels have been sluggish, and he has noted that the stools have been lighter in color during the last few days. His skin has had a yellowish tint for several days.

Previous Diseases.—The patient has had very few illnesses; until seven years ago his health was very good. Twelve years ago he contracted a preputial sore, which was not thoroughly treated. Eighteen years ago he had a neisserian infection.

Family History.—His father died at eighty-two with organic heart disease; his mother died at fifty-four, cause unknown. He has 6 brothers and 5 sisters living and well.

Habits.—His habits are sedentary. He does not use alcohol, but uses coffee freely and smokes excessively.

Examination.—The patient is moderately well nourished, but has a look of pain upon his face, and seems very much worried. The skin is yellow and likewise the scleræ. The reflexes are all intact. The eyes react to light and accommodation in a normal manner. The lung findings are normal. The heart is slightly enlarged; the apex is just outside the nipple line, the second aortic tone is accentuated; there are no murmurs to be heard; the action is slow and regular.

The blood-pressure measures 148 systolic and 78 diastolic.

The right upper quadrant of the abdomen is very tender. The liver descends 4 inches below the costal margin in the mammary line. It is tender, but there are no definite evidences of irregularity. There is no evidence of ascites. The spleen can be palpated one fingerbreadth below the costal arch. It is firm, but not tender.

The urine shows a trace of albumin and a few hyaline casts. It contains bile.

The stools show a clay color, with an increased amount of fat; there is no occult blood.

The stomach test shows normal findings. There is no occult blood present.

The blood shows a picture of secondary anemia of moderate grade. The Wassermann reaction is positive.

x-Ray examination shows normal lung findings. The heart is enlarged, especially to the left, and there is a diffuse dilatation of the aorta of moderate grade.

The bismuth examination of the gastro-intestinal tract shows the stomach negative, the duodenal bulb negative, the colon moderately spastic, no tenderness of the cecum; the appendix could not be seen. There was marked tenderness in the right upper quadrant over the liver and gall-bladder.

This patient undoubtedly has a disease of the liver and

probably of the gall-bladder. If he had not had a definite history of a specific infection, and if his blood at that time had not shown the Wassermann reaction to be positive, I should certainly have considered surgical intervention, but because of the above findings I thought it wise to put the patient upon specific management first to see what could be accomplished by such therapy.

I have kept him under observation, and during this time he has received the usual treatment with injections of mercury and salvarsan. From the outset he has shown improvement, until now his liver is not tender, it has grown smaller, his clinical symptoms are gone, he feels very much better. I shall watch him from time to time and give him specific therapy whenever his clinical symptoms or the blood Wassermann test indicate the need for it.

Case II.—The next patient, Mrs. J. L. F., aged forty-one years, first consulted me in July, 1917. I have had her under observation since that time, and will give you the clinical history and findings from the outset, and tell you of her subsequent course.

In 1917 she complained of pain in the right upper quadrant of the abdomen, of nausea and vomiting, of headache and of jaundice, and stated that for several years she had not been feeling well. Several months before consulting me she began to have abdominal pain which was located in the right upper quadrant, near the costal margin, which occasionally was referred to the back or upward to the shoulder. In between severe attacks there was a constant dull ache and she was conscious of a discomfort all the time. The attacks have been coming at intervals of one or two weeks and have been associated with nausea and vomiting. One week ago she noticed that her skin was yellow and since that time she has had malaise and headache along with constant pain. She does not know of any previous illness except the diseases of childhood. She has not had any eruption or the usual signs of secondaries except for a sore throat, which she says has bothered her from time to time for several years.

Menstrual History.—Menstruation began at thirteen years, and has been regular. She has not menstruated for the past few months; there has never been any pregnancy.

Family History.—Her mother died of puerperal sepsis; her father died of heart disease. She has one brother and one sister living and well.

As you observe, this patient has a deep, generalized icterus. She is apathetic and looks weak, although she is moderately well nourished. Her eyes react promptly to light and the reflexes are intact. There is no adenopathy, the lungs are normal, the heart shows a slight enlargement to the left and right. There is a systolic murmur heard over the base and transmitted into the vessels of the neck. The second aortic sound is accentuated; the heart action is regular and slow.

The blood-pressure measures 160 systolic, 90 diastolic.

The liver extends three fingerbreadths below the costal margin; it is distinctly tender and seems to be slightly irregular. The tenderness is most marked in the region of the gall-bladder, but the gall-bladder cannot be palpated.

There are no abnormal findings in the nervous system.

The tonsils are enlarged and reddened, but there are no patches to be found in the mouth.

The urine showed no abnormal findings except for bile, which was present in large quantities.

The stool shows the typical clay color, but does not contain any occult blood.

The stomach test shows a free acid of 48 and a total acidity of 80; no blood is present, the ferments are present in normal amount, and the motility of the stomach is normal.

The blood shows a secondary anemia of slight grade. The blood Wassermann reaction is markedly positive.

The *x*-ray examination shows normal lung findings. The heart is slightly enlarged; there are no signs of aneurysm.

The bismuth examination of the stomach and duodenal bulb are both normal. There is no evidence of trouble in the colon.

There is marked tenderness over the region of the liver and the liver shadow is increased in size.

This patient has been observed from time to time since July, 1917. In her case, as in the case of the previous patient, because of the finding of a strongly positive Wassermann reaction I decided to try specific therapy before having surgical aid. After daily injections of mercury and weekly injections of salvarsan and a plain diet the patient began to show rapid signs of improvement, and she left the hospital at the end of two months in good health. At intervals since that time she has received a course of mercurial injections along with salvarsan, and her health has been maintained for the past three and a half years without any of the previous abdominal symptoms. Although this patient does not know of her luetic infection, I have been able to learn through her husband that he had a definite infection and has been under treatment for a number of years.

In both of these cases, without evidence of syphilis, one would be justified in having an operation carried out for disease of the gall-bladder, for the clinical picture and findings spoke definitely for gall-bladder disease, probably associated with gall-stones, and both of them showed signs of common duct obstruction. Although I cannot state that in these cases gall-stones are absent, yet the cases justify the plan of treatment which I have followed in these and similar ones for some years. I am sure that it is proper in cases that have definite evidence of syphilis who have not had active, thorough treatment, to temporize before surgical aid is sought when symptoms of the type exhibited by these patients present themselves. If, after several weeks' effort, the patient does not show definite improvement, then surgical aid should be sought in addition to the medical active treatment.

I wish to emphasize that syphilis of the liver may at times give a picture resembling gall-stones so closely that clinicians of wide experience have difficulty in deciding the question of the proper course to pursue along medical or surgical lines. It is always wise to keep this possibility in mind in all cases with the picture of gall-stones. When the blood is negative for the Wassermann test if there is any suspicion at all of the case being atypical it is wise to study the spinal fluid before definitely excluding syphilis.

I have 2 more patients that I want to present to you, in both of whom there was evidence of intestinal trouble which was of such a nature that I advised operation for a tumor of the colon.

Case III.—The first patient, Mr. G., fifty-seven years old, came under my observation November 10, 1919. He was complaining of pain which was associated with belching and followed by nausea and vomiting, which brought temporary relief. The pain was felt in the epigastrium and came on two or three hours after eating. The pain was aggravated by taking sour substances and relieved by alkalies. These attacks had been present for the last year, and during that time he has had five attacks, and has been given hypodermic injections to relieve the pain. The vomitus was clear and did not show any blood. He has never been jaundiced. The stools have always been normal in color. Although his stomach does not give him severe trouble in between attacks, yet he says he belches after every meal and at times feels considerable distress or discomfort in the region of his stomach. During the attacks he has manifestations of vertigo which are not present in the intervals.

Previous Diseases.—He has not had any illnesses, having had remarkably good health since childhood.

Family History.—His father died at the age of seventy-five; his mother is living and well. He has 3 brothers and 2 sisters, all in good health.

Physical Examination.—Examination reveals some emaciation and he looks pale. His reflexes are intact. His eyes react promptly to light and accommodation. There are no abnormal findings in the lungs and the heart findings are normal. The liver extends three fingers below the costal arch; it is not tender, it is smooth, and of normal consistency. There is tenderness just below the liver on the right, but no definite mass can be felt. There is no tenderness in the region of the appendix. He has small hemorrhoids. There was no mass to be felt in the rectum and the prostate is normal.

Laboratory Findings.—The urine shows normal findings. The stool shows a markedly positive test for occult blood; there is, however, no evidence of macroscopic blood, and the stool is

greenish-brown in color. The stomach test shows normal motility and the acid titrations are normal. There is no occult blood present.

The red cell count is 3,250,000, the white cells 10,500; the hemoglobin 58 per cent. The differential leukocyte count shows 18 per cent. small mononuclears, 2 per cent. large mononuclears; 75 per cent. polymorphonuclear neutrophils; 3 per cent. eosinophils; 2 transitional cells. The blood-smears show the picture found in secondary anemia. The blood Wassermann test was negative.

x-Ray examination made by Dr. E. L. Jenkinson at this hospital shows a normal esophagus. The stomach is normal in size and shape, no filling defects or obstruction on the greater curvature, the waves normal, and the pylorus regular. The duodenal bulb shows an irregularity at the apex. The stomach empties in four and a half hours. The large bowel is flat throughout, the appendix is flat, movable, and not tender, the cecum and ascending colon are freely movable.

The conclusion from the *x*-ray findings was that of duodenal ulcer, and this seemed more likely from the finding of blood in the stool on a meat-free diet. The patient was put on ulcer management for a week, but the bleeding continued, and in spite of the negative *x*-ray evidence and because of the man's emaciation, I felt that we were probably dealing with a tumor of malignant nature and advised operation. Dr. L. L. McArthur, at operation, found a small tumor at the hepatic flexure of the colon. Because of the anemia and the poor condition of the patient a two-stage operation was performed; at first an anastomosis of the ileum to the transverse colon was made, and a few weeks later a complete excision of the entire ascending colon and one-third of the transverse colon was done. The tumor was of the annular type and its surface was ulcerated. It was attached to the duodenal bulb (Fig. 266) and had to be separated from it. The microscopic sections showed typical findings of carcinoma.

The reason that the tumor, which was small in size and located at the hepatic flexure, could not be demonstrated by the *x*-ray.

was probably due to the fact that it was located in a fold of the colon and at the hepatic flexure and was obscured by it. Possibly a repetition of the *x*-ray examination would have shown some filling defect in this region. The fact that the tumor was adherent to the duodenal bulb and had to be separated from it accounts for the duodenal bulb deformity.

Fig. 266.—Case III, Mr. G. Tumor of the cecum.

I advised this patient to have surgical intervention because on medical ulcer management the bleeding continued and, as I have mentioned before, I felt that we were not dealing with an ordinary ulcer. The patient since his operation has been gaining rapidly, and I am in hopes that there will be no recurrence of the malignant tumor of the hepatic flexure of the colon, which was removed.

Case IV.—The next patient, Mr. H., aged sixty-one, consulted me several months ago complaining of epigastric distress, loss of strength, and anorexia. At that time he stated that he had not had his usual appetite for several months, but did not have any pain, nausea, or vomiting. He would waken in the early morning with an indefinite distress, but could not definitely locate or define it. His bowels had a tendency toward constipation; he had not passed any dark stools to his knowledge.

Previous Illnesses.—In his previous history he states that he had an attack of influenza during the epidemic thirty years ago, that he had an operation for hemorrhoids three years ago, and that his appendix was removed eight years ago.

Family History.—His father died at the age of seventy-two, of a sarcoma; his mother died at fifty-eight, cause unknown.

Physical Examination.—The patient when first seen showed a slight cyanosis, especially marked in the lips, but seemed well nourished. There were no abnormal findings in the nervous system. His eyes were normal in all reactions. There was a slight emphysema of the lungs, but otherwise his lung findings were negative. The heart showed some enlargement to the left and right, the apex being found 1 inch to the left of the nipple line. There was a systolic murmur heard all over the heart area and transmitted into the vessels of the neck. There was a definite arhythmia present.

The blood-pressure was 125 systolic, 73 diastolic.

The liver extended 4 inches below the costal margin; it was smooth and firm and not tender. The spleen could not be palpated. There was tenderness in the epigastrium, but no mass could be felt. There was also present an inguinal hernia of small size on each side. The prostate was slightly enlarged, but not tender.

Laboratory Findings.—The urine showed a trace of albumin, with a few hyaline casts, but was otherwise negative. The stool showed definite evidence of occult blood. The stomach test on repeated examination, including the fractional method, did not show any free hydrochloric acid at any time. There was no blood present in the stomach contents.

The blood examination showed 3,984,000 red cells, 9500 white cells, and a hemoglobin of 62 per cent. The differential leukocyte count showed 23 per cent. small mononuclears, 2 per cent. large mononuclears, 69 per cent. polymorphonuclear neutrophils, 1 eosinophil, 1 basophil, and 4 transitional cells. The

Fig. 267.—Case IV, Mr. H. Tumor of the cecum.

smears, like those in the preceding case, showed the picture found in secondary anemia. The blood Wassermann test was negative.

The *x*-ray examination made by Dr. E. L. Jenkinson at this hospital showed normal findings of the lungs; the heart showed moderate enlargement to the left and right; the aorta showed

slight diffuse dilatation; the stomach showed early emptying, there was no defect seen, and the duodenal bulb was negative.

The colon enema gave the following findings: The rectum and sigmoid filled rapidly and showed no defect; the transverse colon was spastic, especially near the splenic flexure; the hepatic flexure was normal. The part near the cecum could not be filled out and showed a filling defect involving the outer and middle part of the tip of the cecum (Fig. 267).

Because of the filling defect at the cecum and the blood in the stool it was decided that this patient had a tumor of the cecum. He had had his appendix removed eight years ago, and it was necessary to exclude a deformity due to a scar following the operation. In spite of the absence of the usual obstructive symptoms of a tumor of the cecum, it was decide at the persistent presence of occult blood in the stool and the *x* ray deformity of the cecum probably spoke for a tumor, and operation was advised. Dr. L. L. McArthur resected the terminal ileum and nearly all of the ascending colon and then did a lateral anastomosis. On the inner surface of the cecum at the site of the old appendectomy scar a small cauliflower growth was found. Microscopic sections showed the typical picture of carcinoma.

This patient, again, was puzzling for a time to decide the question of surgical intervention. He had practically no clinical evidence of tumor of the colon, as in the previous case, and in both of these cases it was a question of deciding the proper course to pursue from the laboratory and *x*-ray findings plus the clinical judgment based on the condition of the patient.

At the time of operation there was no evidence of metastases, and I hope that none will develop. The further management of both these cases is properly one of care in diet and proper hygiene to recover from their anemia and to regain strength. I feel that the outlook for both of them is decidedly good.

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THE MEDICAL CLINICS OF NORTH AMERICA

VOLUME 3
1919—1920

PHILADELPHIA AND LONDON
W. B. SAUNDERS COMPANY

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PUBLISHED BI-MONTHLY (SIX NUMBERS A YEAR), BY W. B. SAUNDERS COMPANY, WEST WASHINGTON SQUARE, PHILADELPHIA

PRINTED IN AMERICA

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